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Dynamics and Management of Self-Induced Eruptions

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• **Self-induced eruptions are always expressions of an emotionally disturbed person. They cover a wide variety of injuries and include aggravation of preexisting dermatoses, neurotic excoriations, mucocutaneous changes from compulsive movements, factitial dermatitis and trichotillomania. The emotional disturbances in such patients range from mild psychoneuroses to severe psychoses. Attention should be focused primarily on the emotional disturbance. The management of psychotic patients is the domain of the psychiatrist. But those unsuitable for psychiatric care and persons with mild psychoneuroses—who fortunately constitute the majority among patients with self-inflicted eruptions—should remain with their dermatologist or general physician and get from him effective supportive psychotherapy in addition to such treatment for the self-induced damage to the skin as may be indicated.**

THE VARIOUS KINDS OF true dermatoneurosis may be classified as: Erythrophobia (abnormal blushing); hyperhidrosis; phobias; self-induced eruptions; trichotillomania; delusion of parasitosis; dermatologic hypochondriasis; stigmatization.

For discussion with regard to dynamics and the management of patients with these conditions, I have chosen in particular the self-induced eruptions, trichotillomania and delusion of parasitosis. For a clearer understanding of what management involves, I will give first a brief definition and illustrations of the clinical entities from the dermatologist's point of view.

I have found it convenient to classify the self-induced eruptions as follows[3]: Aggravation of preexisting dermatosis; neurotic excoriations; mucocutaneous changes owing to compulsive movements; factitial dermatitis.

AGGRAVATION OF PREEXISTING DERMATOSIS

The observation that patients with psychocutaneous excoriation syndromes tend to express aggressiveness more masochistically than others[5] and the preponderance of the anxious obsessional personality trait in patients with cutaneous disease[2] account for the frequently noted self-induced aggravation of dermatosis in such patients. The obsessional patient is overconscientious and preoccupied with cleanliness, order and routine. He makes a ritual of the prescribed therapeutic measures; thus he is apt to overtreat himself with resulting aggravation of the dermatosis. This is especially true of the patient with neurodermatitis. But the obsessional trait is shared by many persons who have cutaneous disease; self-induced aggravation is commonly encountered in inflammatory chronic dermatosis not ordinarily thought to be associated with psychic factors. Therefore the obsessional trait and the neurosis of which it is a symptom are of considerable importance in contact dermatitis, especially the occupational variety.

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Overtreatment and rubbing usually is rationalized by the patient as a necessary part of therapy.

NEUROTIC EXCORIATIONS

A more severe form is termed neurotic excoriations, although there is no fixed point at which simple self-aggravation ends and neurotic excoriation begins. There are many transitional forms. The varying degrees of excoriation in acne vulgaris, from slight to severe, are illustrative. In this common disease of the adolescent, aggravation of the blemish occurs because of constant squeezing, pricking and chin-fondling of the self-conscious patient. In cases in which the disease actively continues beyond adolescence, the inflicted damage is sometimes more severe, resulting in unsightly scars.

The most typical lesions are produced by the patient with his fingernails because of intolerable itching or burning or an uncontrollable desire to dig into the skin in certain regions; the usual sites are the extensor surfaces of the arms and forearms, the thighs and legs, the face, and the upper part of the back.

The excoriations, most often about 0.5 cm. in diameter, are covered with sanguineous crusts and surrounded by an erythematous border. The crust eventually falls off, leaving a pink scar—which later becomes pale—with a hyperpigmented border. Lesions in all phases of development are present simultaneously. Neurotic excoriation should not be confused with factitial dermatitis; in the latter the patients will not admit to having produced the lesions unless confronted with irrefutable evidence, while those with neurotic excoriations readily admit what they have done but add that they simply cannot help digging at the skin because of intolerable discomfort.

To the patient the important feature is discovery of an irregularity of his skin, a "core," a "bump" or a "pimple" which he is compelled to remove; often follicular plugs are the object. If he accomplishes removal, he experiences relief and satisfaction. Minimal as may be the cutaneous changes that preoccupy the patient, there always is some slight abnormality of the skin to which his neurosis becomes fixed.

The average patient with neurotic excoriations does most of his digging into the skin before going to bed, and spends as much time—a few minutes to several hours—in the performance as is required for relief of his tension. Although aware of the irrational and self-destructive nature of his behavior, he is unable to discontinue the habit for long. On voluntary attempt to stop this harmful practice, he builds up tension to an intolerable point and is driven back to the self-damaging activity. The tension and discomfort are similar to that suffered by obsessive-compulsive patients when unable to complete their rituals or ceremonials.

A complex pattern of reactions underlies this symptom rather than a single neurosis or psychosis. It is felt that these cutaneous manifestations are end results of a specific type of compulsive behavior, the origins of which lie in early fears and unexpressed rage toward a prohibitive and punitive parent. The self-inflicted lesions represent a variety of emotional disturbances: repressed rage, vindictiveness and hostility toward a childhood authority and later to the frustrating outside world; guilty fear, self-punishment, and self-destruction for attempting to reject that authority; finally, riddance of dirt, excreta, smegma, and any form of cutaneous disfigurement, the latter representing contamination, badness, and a form of punishment for transgression. The extent of the cutaneous lesions and the degree of self-destruction are in proportion to the distortion of the personality.

MUCOCUTANEOUS CHANGES FROM COMPULSIVE MOVEMENTS

Injury to the skin from compulsive movements is well known to occur in animals. "Wolf-biters" among dogs and "tail-chewers" among monkeys are examples. Nailbiting and knuckle-biting, the latter leading to the formation of the so-called knuckle pads, are commonly encountered in persons of all ages.

The biting, chewing or sucking habit always has an erotic component. The elongated shape or leathery consistency of one of the small labia on the vulva, the thickening of the clitoris and of the tissue surrounding it, as well as similar changes of the perineum in persons of both sexes, are eloquent manifestations of masturbatory practices. Thickening of the mucocutaneous tissue of the lips or tongue in the form of a circumscribed spot as a result of compulsive manipulation has given rise to confusion with leukoplakia. It is not uncommon to find cutaneous changes in feebleminded persons who have the habit of frequently biting themselves with a violent chewing motion on the forearms, hands and fingers, resulting in areas of thickening, hyperpigmentation and hypertrichosis. It is believed that the hypertrichosis is produced by the intermittent pressure associated with friction.

Compulsive movements may lead also to development of contact dermatitis—for example, nickel dermatitis of the lower lip produced by the patient's rubbing her wedding ring against that part of her face.

FACTITIAL DERMATITIS

The term *factitial dermatitis* is loosely applied to any kind of self-inflicted injury to the skin. A preferred usage limits it to cases in which the patient

consciously produces the artifacts but denies his responsibility for them, the denial distinguishing the disorder from neurotic excoriations.

Diagnosis is determined by the character of the lesions and by exclusion of the kinds of dermatosis with established characteristics. The lesions usually are in a region readily reached by the right hand (or, in left-handed patients, the left hand). Their artificial appearance arouses suspicion; the pattern is distinctive and bizarre, and differs from that encountered in other kinds of dermatosis—features immediately apparent to the expert. While the changes may be due to a chemical or thermal burn, they frequently are of other traumatic origin. Cutaneous artifacts produced by an instrument often are of regular—even geometric—arrangement, a feature most uncommon in spontaneous eruptions. Chemical burns, likewise, frequently are of a regular pattern: A liquid may produce streaks so that linear extensions of the main lesion often are seen, as would result were a drop of a chemical to run downward on the surface. Sudden appearance overnight of a full-blown lesion or lesions should arouse suspicion of factitial eruption.

When the suspected lesions are located on the extremities, application of a simple Unna's zinc-lime gelatin or other occlusive dressing—if the eruption is self-produced—will prevent further infliction and permit preexisting lesions to heal in the protected areas.

However, differential diagnosis from spontaneous inflammatory dermatosis is at times exceedingly difficult.

The psychic importance of factitial dermatitis is that although the patient consciously produces the lesions, he is not aware of the basic reasons for this self-destructive behavior. Such persons always deny any part in causing the eruption unless they are caught in the act. Factitial lesions usually are in a prominently exposed area, psychologically chosen to enable the patient to obtain attention, sympathy, pity or compensation, or to use as a device for escaping responsibility.

The patient who presents factitial dermatitis is, inherently, unable to face or admit inadequacy. The lesions serve to rationalize his belief that he has failed to achieve success in life. The majority of patients seen with this condition are persons of middle age who either have not attained their goals or have slipped far below their set standards, socially and economically; they have given up the competitive struggle; they save face by having a physical illness.

Psychiatric studies have shown that patients with factitial dermatitis are of low intellectual level and therefore—in contrast to patients with neurotic excoriations—rarely amenable to accepting psychiatric help, even if it is offered. Most observers agree that it is bad therapy to unmask those who perpetrate self-infliction without first encouraging them and building them up psychologically. These patients are unaware of the motivation of their self-destructive behavior; sudden exposure may deprive them of protective covering at a crucial age when they are unable to meet competition. Because of their emotional immaturity, deep psychotherapy may not be indicated in such patients.

TRICHOTILLOMANIA

Trichotillomania is the abnormal and uncontrollable desire to pull out one's own hair. The patients are predominantly children, and the condition probably is more common during early childhood than is generally recognized. So many hairs may be pulled out that some areas may be virtually denuded, or the hairs may appear to be broken off. The scalp and the eyebrows are most frequently attacked, although the pubic hair is not exempt. Subjective symptoms are usually absent, but intense pruritus has been reported. The artificial appearance and irregularity of the bare or sparsely covered patches aids diagnosis.

The psychiatric mechanism appears to differ according to the age groups and the level of the emotional disturbance. In neurotic children trichotillomania may represent anger against a rejecting parent which has been turned against the self.

Trichotillomania of the eyebrows, eyelashes or pubes may occur as a mild neurotic symptom, however. The patients fondle, smell, chew or bite the extracted hairs, or roll them on the tongue and experience sensual pleasure in doing so; in some instances it may be considered a masturbatory substitute. Trichotillomania of this type usually subsides as the patient becomes able to work out his psychologic problem.

Conflicts in the sexual sphere and feelings of guilt appear to be predominant in patients with this manifestation.

In adults severe trichotillomania is often associated with schizophrenia and other forms of psychosis. The prognosis depends entirely on the background of the case. In schizoid, paranoid or highly narcissistic persons it is poor, while a good prognosis is the rule in patients whose emotional development is more mature.

DELUSION OF PARASITOSIS

The delusion of parasitosis is a mental disorder characterized by an unshakable false belief that live organisms, such as mites or insects, are present in the skin.

In some patients demonstrable changes in the skin do not occur; in others, typical neurotic excoria-

tions are present. In elderly patients the disorder sometimes is associated with senile pruritus. The principal complaints are itching, biting, stinging, burning and crawling sensations. The patient may produce "evidence"—small objects which he claims to have rubbed off or dug out of his skin, consisting usually of epidermal debris or bits of lint from the clothing, but occasionally a bottle filled with ants, small beetles or other insects that obviously could never have come from within the skin. Various modes of extraction are used by the patients to rid themselves of these supposed parasites—digging with the fingernails, needles, pins, tweezers, scissors, scalpels and knives, plus sterilization with various antiseptics and caustics. As a result of all this excoriation and self-destruction of tissues, varying degrees of infection and scarring ensue. In most patients the lesions are noted at first on the exposed parts of the body, particularly the face, neck, and arms; later they are produced on other parts of the body—the chest, axillae, groin and scalp.

The emotional disturbances which underly the symptom of delusion of parasitosis are toxic psychosis, schizophrenia, involutional melancholia and paranoia. When proper psychiatric care is given the prognosis is good in the case of toxic psychosis, poor in schizophrenia and involutional melancholia, and almost hopeless in paranoia. Paranoid patients are not suitable subjects for psychoanalysis. These unfortunate persons should be placated, and treated gently and tolerantly, without trying to reason with them, to confer insight or to change their intrinsic nature. Rather, extrinsic factors, such as environmental influences, should be manipulated to avoid areas of conflict for the patient.

At times the mental disturbance of pellagra takes the form of delusion of parasitosis and disappears with appropriate therapy. This is important because many patients with this delusion have deficient diets. Investigation of dietary habits is always indicated.

Occasionally, patients are observed in whom the fear of infestation proves to be a phobia and not a delusion. The distinction is important. Phobic patients have a persistent fear of contamination, although after being presented with laboratory proof of its fallacy they are able to recognize the irrational nature of their fear. However, because the fear is based on unconscious conflicts and is therefore not amenable to logical argument, it persists in the same or a different form. The patient with a delusion, on the other hand, remains convinced, regardless of proof, that his fears are justified. Psychotic processes, to which delusions are related, are based on a regression to a very early stage of psychosexual development, when the infant is unable to discriminate between reality and fantasy. Phobias are related to a much later stage of infantile development, when the function of reality testing is much more firmly established. Hence the prognosis of a patient with a phobia is infinitely better than that of one with delusion.

All patients who appear to have a delusion of parasitosis are entitled to a routine search for parasites and treatment for any dermatosis that may be present. The absolute necessity for painstaking examination is well exemplified by reports of seemingly deluded patients who were found to be actually infested with lice or mites.

TREATMENT

In order to avoid waste of time and effort, it is necessary to decide whether it will be best for the patient that he remain with you or be referred to a psychiatrist. In answering this question I use two criteria: The degree of his emotional disturbance and his suitability for psychiatric care.

If signs of steadily deepening depression or symptoms of psychosis are evident, I refer the patient without delay. Occasionally a psychotic patient may be encountered among those with any of the previously discussed cutaneous diseases, but the incidence of psychosis is higher among those with either factitial dermatitis or delusion of parasitosis.

If the patient is deeply disturbed but not obviously psychotic, I base the decision for referral on his suitability for psychiatric care. A good candidate for psychotherapy recognizes and accepts the causal role of emotional disturbance. If he denies it and his attitude toward psychiatric concepts is hostile an attempted referral would only result in the loss of the patient.

This leaves the majority of patients with self-induced eruptions to our own combined dermatologic and psychotherapeutic management. I have frequently observed that a dermatologist, in carrying out this combined task, is apt to underrate his ability to give effective supportive psychotherapy. None of us should or would assume the role of psychiatrist without special training in that field. I would not dream of exploring the deeper layers of the patient's unconscious or uncovering repressed conflicts associated with his early life. If I were to do this I might find myself in the undesirable role of Goethe's Sorcerer's Apprentice, who found himself unable to control the forces he had unleashed.

Our main task, as I see it, is to make clear to the patient that we understand him and are sympathetic to his problem. If good rapport has been established during the first visit—and in my experience that first interview is crucial—I usually make some final reassuring comments to the patient. For example, to a psychoneurotic woman with neurotic excoriations I might say: "We both are aware that you are producing the sores yourself. We don't

know yet the reason for your doing this but we shall try to find out. I think that I have given you today some food for thought. Go over our discussion in your mind and tell me, next time, any new thoughts you may have on the subject. If you are able to refrain from digging, that would naturally be desirable, but if you are not, go ahead and dig. You understand now that you are doing this to relieve tensions. Perhaps I shall be able to help you to find relief from them without destroying your skin."

When she returns I listen patiently, without interrupting, then discuss her current emotional problems without giving psychiatric interpretations of her anxieties. My weapon is the building of her ego by expressing confidence in her ability to help herself and by aiding her to deal with tensions consciously which formerly had been discharged through a behavior pattern of which she was not fully aware.

I have set myself no fixed time limit in interviewing patients. For the initial visit, because of its all-importance, I always allow at least half an hour. For patients who are sent in consultation, hence with forewarning of the likelihood of psychic factors, I allow a full hour. The time spent on subsequent visits depends on the pressure of work on that particular day, but I always try to reserve at least 15 minutes for discussion.

One must keep in mind that many of these patients are extremely sensitive and proud. While they seem to long for help and guidance, they are outraged if they discern signs of condescension.

I often find it desirable to interview the spouse or the parents to help to orient them toward the patient's problems.

In management, I distinguish between patients whose relative intelligence, basic education and capacity for insight are good and those who are deficient in these qualities. To the former I give no dermatologic therapy because that would only impede the development of insight into the purely emotional nature of their problems. For the latter some form of dermatologic treatment is essential or they will not return. I am aware of the placebo nature of such management, but I reason that it is justified so long as the main emphasis is given not to the dermatologic procedures but to the accompanying reassuring discussion. This holds especially for the elderly patient with delusion of parasitosis for whom psychiatric referral is out of the question for economic as well as medical reasons. Such a patient—usually a lone person with an inadequate diet—will profit from the injections of crude liver extract which he thinks are the real reason for his office visit.

When I feel that sedation is indicated I use it freely but never to the point of turning the patient into a vegetating animal, nor do I force it on him if he is prejudiced against "nerve medicine." Small but steady doses of meprobamate seem to serve best.

As to the value of rest, including hospitalization —which I used to espouse in the management of the neurodermatoses[11]—I have changed my mind. I have come to the same conclusions as Seitz[4] who said that nervous fatigue is not a cause of psychocutaneous disorders. Overwork is a symptom, not a cause, of chronic unresolved emotional tension. Work is salutary because it helps to relieve tension, while enforced idleness has the opposite effect. But encouraging the patient to outline a regimen that will provide for adequate sleeping hours and, if possible, a daily nap, and a sensible balance between work and play is of course highly desirable. A relaxing hobby should be active and manual, and not passive-intellectual, such as reading. Self-inflictions—to take again neurotic excoriations, as an example—are essentially tactile conditioned reflexes and it is therefore logical that the substitute should be a tactile activity. Knitting serves well for female patients, both teenagers and adults, with excoriated acne; ceramics or sculpturing are desirable substitutes for adults.

As to the results of treatment I would say that they naturally vary a great deal. Some of my patients with neurotic excoriations have stopped digging into their skins after the first interview and have remained well. I have had others who continued their self-inflictions for several months and then stopped. And I have treated others who continued to abuse their skins right up to the time of involuntary parting, and who probably still dig, under somebody else's auspices; but they are relatively few. Improvement is the rule. Even the little old lady with delusion of parasitosis who cheerfully answers my question, "And how are the bugs today?" by saying "Only a few feelers in the ears, but I got them out by the root," thus acknowledges relief obtained.

Treatment of patients with self-inflicted lesions is an intuitive and time-consuming task. Failures are inevitable. But often our efforts are successful.

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Head Injuries in Infants and Young Children

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• **Head injury in infants and young children may produce lesions that are relatively unique for this age group. The uniqueness is generally due to the structural immaturity of the skull, meninges and brain.**

"Derby-hat" and diastatic fractures are common in this age group. Spurious meningoceles result from tearing of the dura which is closely adherent to the skull. The syndrome of "delayed" concussion is more commonly manifested in children. Extradural and subdural hemorrhage may develop from lacerations of the major venous sinuses. A classical extradural hematoma may occur in the absence of fracture across meningeal arterial channels.

The management of patients with head injury has been improved by the more frequent use of tracheotomy, hypothermic techniques and drugs of the "lytic cocktail." Solutions of urea in 10 per cent invert sugar are administered intravenously to control cerebral edema in selected patients.

Head injuries in infants and children of pre-school age have many unique features that distinguish them from those occurring in older children and adults. These differences are due largely to the structural qualities of the skull and the immaturity of the central nervous system in this age group. This report will deal with those facets of the head injury problem that are of greatest importance to the pediatrician. Particular stress will be placed on the management of "closed" injuries not requiring surgical treatment.

ANATOMICAL AND PATHOLOGICAL CONSIDERATIONS

Scalp Injuries

The abundant blood supply and inability of the blood vessels to contract or constrict allows scalp lacerations to bleed more profusely than comparable wounds in other body areas. In infants and small children the loss of blood may produce shock and necessitate emergency replacement by transfusion. Scalp lacerations usually heal by first intention if properly cleansed, debrided and sutured.

Subgaleal hematomas are often perplexing, inasmuch as their soft centers and hard, raised edges may give the impression of an underlying depression of the skull. These hematomas must be distinguished from "spurious meningoceles," which are subgaleal collections of cerebrospinal fluid. The latter are due to an underlying fracture of the skull, accompanied by dural and arachnoidal tears that permit the extracranial escape of cerebrospinal fluid. Both of these collections usually disappear with the passage of time. At times, needle aspiration may be required.

Skull Fractures

In contrast to the skull of the older child and adult, which is relatively rigid and nonyielding, the skull of the infant is elastic and subject to considerable deformation. Its vault consists of rather loosely joined flat bones separated by sutures and fontanelles. As compared with the elastic vault, the base of the skull is relatively rigid. The posterior fontanelle is usually closed at the age of two months, the anterior fontanelle between the ninth and sixteenth month. The sagittal, coronal, lambdoidal and temporosquamosal sutures are not firmly united until the child is about four years of age.

Due to the elasticity of the vault of the infant's skull, depression of bone may occur without extensive fracturing, the fracture being of the so-called "ping-pong ball" or "derby-hat" type. Because of the loose joint at the suture lines, diastatic fractures are more common in children. The infant dura is rather rigidly attached to the inner table of the skull and is, therefore, commonly torn directly beneath a fracture line. As noted previously, this often permits the escape of cerebrospinal fluid beneath the scalp, forming the so-called spurious meningocele. Dural tears involving the major venous sinuses may produce subdural and extradural hematomas.

The more serious skull fractures are those in which there is comminution of bone. These comminuted fragments are frequently driven inward, producing a simple, depressed fracture or are associated with an overlying laceration of the scalp resulting in a compound, comminuted, depressed fracture. At times, the in-driven fragments will penetrate the dura and lacerate the underlying brain.

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In general, linear fractures in infants and children do not, per se, present a problem. Unless the edges are widely separated, most linear fractures heal quickly and are no longer apparent on radiological examination in three to six months. If the fracture line crosses an arterial or venous channel, the child should be closely observed for signs of increasing intracranial pressure that signal the formation of a subdural or extradural hematoma.

Linear fractures involving the base of the skull (basal fractures) may not be apparent on either clinical or radiological examination. The diagnosis is usually established by finding leakage of cerebrospinal fluid from the nose or ear. Fractures involving the paranasal sinuses may enable air to enter the intracranial chamber and produce a pneumocele or spontaneous ventriculogram. The presence of rhinorrhea, otorrhea or intracranial air converts a simple fracture into a compound fracture and requires appropriate antibacterial prophylactic therapy.

Brain Injury

Concussion, cerebral contusion and laceration, cerebral hemorrhage and edema, and meningeal hemorrhage in its various forms are common sequelae of cranial trauma in both children and adults. However, because more of the energy of the blow is absorbed by the deformation of the skull, the brain is relatively more protected from injury in infants than in adults. Some authorities point out that for a similar reason the brain stem of infants is less vulnerable.

The most common change in the human brain resulting from a head injury is the phenomenon known as concussion. Before World War II, concussion was considered to be a mysterious disturbance of cellular function which was unaccompanied by any demonstrable pathological change in nerve cells. However, the stimulus provided by the war led many investigators to reassess the brain injury problem, particularly the nature of concussion. It has now been shown that concussion produces swelling in nerve cells, chiefly those of the brain stem's reticular formation. Intensive studies of the function of the reticular formation have disclosed that this area has a modulating and integrating effect on motor and sensory function, autonomic balance, visceral function, alertnesss and sleep and the maintenance of consciousness. The diagnosis of concussion is established if there has been a disturbance of consciousness following a head injury. This may vary from momentary confusion to coma. Children with relatively minor head injuries may have what has been termed delayed concussion. This is characterized by drowsiness, pallor, vomiting and diaphoresis, coming on several hours after the injury. In the presence of these symptoms, close observation for signs of intracranial bleeding is required even though most often the child recovers promptly. How this delayed form comes about is obscure.

Frequently, the forces involved in cranial trauma will produce gross lesions in the central nervous system, such as contusion and laceration. In severe closed head injury, the frontal and temporal poles of the brain are subject to the greatest damage. This is due to shear strains that develop in these lobes because of the rotation of the brain within the skull at the time of impact. Cerebral contusion and laceration may occur directly beneath the site of the blow (*coup* injury) or at a point directly opposite to it (*contrecoup* injury).

Edema of the brain will develop in the wounded areas. Generalized cerebral contusion may result in such widespread edema that the intracranial pressure will reach critical or fatal levels. Reduction of this edema is imperative, inasmuch as the associated ischemia may produce widespread death of nerve cells.

Persisting neurological defects will occur as a result of irreparable brain damage. These paralytic phenomena will vary, depending on the location and extent of the injury. Convulsions will develop as a late manifestation in approximately 2 per cent of patients with closed head injury and in 20 per cent of the patients in whom the brain wound is associated with dural penetration.

TREATMENT

Emergency Measures

In the management of the child with a head injury, the four major steps to be taken are: Establishment of an adequate airway followed by the administration of oxygen; control of shock; control of hemorrhage; assessment of the extent of injury.

Establishment of an adequate airway is the first and most important step. Patients with head injury commonly have respiratory obstruction due to depression of the gag, cough and swallowing reflexes coincident with the accumulation of the pharyngeal secretions. During transport to a place of treatment the child should be placed in a modified prone position to promote gravity drainage. If suction is available, a soft rubber or plastic catheter is preferable to a metal device that might contuse the throat. If these measures do not establish a good airway, direct laryngoscopy, bronchoscopy or tracheotomy may be necessary. Neurosurgeons have used tracheotomy with increasing frequency in the past decade.

Shock is uncommon in uncomplicated head injuries and, when present, is usually the result of

intrathoracic or intra-abdominal injury or fractures of long bones. However, particularly in infants and small children, shock may occur as a result of loss of blood. It is good policy to start an intravenous infusion immediately in all seriously injured patients and arrange for blood typing and cross-matching on the assumption that blood replacement may be necessary.

Hemorrhage from scalp wounds may be profuse, particularly if the larger arteries have been lacerated. Bleeding from most scalp lacerations may be controlled by compression dressings owing to the fortuitous anatomical arrangement of a bony backstop afforded by the underlying skull. Hemostats can be applied to bleeding vessels and incorporated in the dressing. Scalp and other wounds should not be sutured until the condition of the child permits this measure.

Having established measures to control respiration, shock and hemorrhage, the physician can then make an assessment of the extent of injury. From the neurological standpoint, evaluation of the state of consciousness is of paramount importance. Disturbed consciousness may vary from confusion to coma. After investigation on this point, the examiner should proceed with a systematic neurological examination, the completeness of which will depend on the responsiveness of the patient. The size, equality or inequality and reaction of the pupils should be noted. Ophthalmoscopic examination is rarely helpful but may disclose acute papilledema or retinal hemorrhage. The ears and nose must be examined for evidence of leakage of cerebrospinal fluid. Seventh nerve involvement may be noted by close observation of facial grimaces. Since neck injury occurs in about 25 per cent of all patients with serious head injury, this area should be carefully examined. The extremities should be observed for signs of weakness or paralysis. Observing the movements of the restless patient often will help in this determination. In a semicomatose patient, movement may be induced by applying a painful stimulus to the hands and feet. The activity of the abdominal and deep tendon reflexes and the plantar responses should be noted. Evaluation of the various sensory functions will depend on the alertness and cooperation of the patient.

Assessment of the neurological status in the immediate post-traumatic period is of paramount importance, inasmuch as it enables the examiner to establish a baseline. A steady improvement in the state of consciousness and the disappearance of abnormal neurological signs is reassuring and usually indicates a good prognosis. Conversely, progressive loss of consciousness accompanied by pupillary inequality, hemiparesis, slowing of the pulse and respiration and an increase in pulse pressure implies that either intracranial hemorrhage or cerebral edema is developing.

Radiological examination is indicated in all patients with head injuries but is not justified as an emergency measure unless it can be done without jeopardizing the patient's chances for recovery. X-ray studies may disclose linear or diastatic fractures which may be of localizing value should an extradural or subdural hematoma develop. The pattern of simple and compound, comminuted, depressed fractures enables the surgeon to plan the operative procedure.

Lumbar puncture is not recommended as a routine diagnostic procedure in patients with head injuries. In performing this test, the attending physician wishes to determine if there is elevation of intracranial pressure and/or blood in the cerebrospinal fluid. Measurements of cerebrospinal fluid pressure are often inaccurate due to the restlessness and straining of the patient or respiratory obstruction or a block in the cerebrospinal fluid pathways. Cerebrospinal fluid blocks may occur at the tentorial notch and the foramen magnum due to shifting of intracranial structures brought about by the pressure of large subdural and extradural hematomas. If this state exists, lumbar puncture may permit further strangulation and hemorrhagic infarction of the brain stem.

In general, lumbar puncture is done to determine whether or not there is blood in the cerebrospinal fluid. Subarachnoid hemorrhage implies that cerebral contusion has occurred, which influences the prognosis and treatment. Patients with meningismus secondary to subarachnoid bleeding may be symptomatically improved by withdrawing 5 to 15 cc. of cerebrospinal fluid at intervals. However, repeated lumbar puncture does not result in a more rapid disappearance of the red blood cells.

TREATMENT

General Measures

The most important part of the program in the management of a patient with a head injury is close observation of the state of consciousness, the vital signs, the size of the pupils and movement of the extremities. The nurses in attendance should be instructed in making these observations. Oxygen and suction equipment should be available at the bedside. Routine and special orders by the physician are outlined in Table 1.

Conscious patients are given clear liquids in whatever amount they wish during the first 24 hours. At the end of this time, solid food and fluids are given as desired. In unconscious patients fluids are administered intravenously in amounts of approximately 10 cc. per pound of body weight per day.

As the state of consciousness improves gavage feedings may be started unless there is a persisting rhinorrhea, serious rhinopharyngeal injury or persistent vomiting associated with depressed pharyngeal reflexes.

Hypertonic intravenous infusions are used only if proven cerebral edema has produced a significant elevation of intracranial pressure. Hypertonic glucose and sucrose solutions are available in most hospitals. Concentrated plasma and serum albumin are effective agents but quite expensive. In recent years, intravenous administration of urea in a 10 per cent solution of invert sugar has been used with increasing frequency. It must be remembered that most of these dehydrating agents have only a temporary effect, which may be followed by a "rebound phenomenon" during which the intracranial pressure may exceed that which existed before they were administered.

Patients with head injuries are usually placed in a position of head elevation to promote venous drainage. However, some neurosurgeons prefer to keep their patients flat. Unconscious patients should have their position changed every two hours to discourage the development of skin and respiratory complications.

Drugs

Analgesics and sedatives that do not significantly impair the state of consciousness or depress respiration may be used. Aspirin may be used freely, and in general it will control headache and restlessness. For more severe pain, hypodermic injection of small amounts of codeine or meperidine (Demerol) may be required. Barbiturates are used to control restlessness. Paraldehyde administered intramuscularly or rectally is probably best for controlling extreme restlessness in children.

Antibacterial therapy is not instituted as a routine measure. It is indicated when there has been extensive wounding with contamination, in semicomatose and comatose patients with depressed pharyngeal reflexes and in patients with cerebrospinal fluid fistulas.

Anticonvulsant measures will be required to combat acute seizures. Barbiturates and hydantoin derivatives are used. It has been our policy to keep all patients with brain wounds on anticonvulsant medication for 12 to 18 months after the injury. If seizures do not develop, the dosage is gradually reduced over this period of time.

Control of Body Temperature

A significant rise of body temperature may occur in patients with cerebral contusion accompanied by hemorrhage and edema. In these damaged areas are nerve cells that will either live or die, depending on the amount of oxygen that is available to sustain their metabolic rate. Reduction of body temperature decreases the oxygen demand and the metabolic rate of these damaged cells, and reduces intracranial pressure by protecting against cerebral edema.

Body temperature of 102° to 103° F. can usually be reduced to normothermic levels by the use of salicylates, alcohol sponges, ice bags, ice-water enemas and the exposure of the patient undressed to air being circulated by a fan. In recent years, more vigorous hypothermic methods have been employed in the management of patients with severe head injury. These patients will often have rectal temperatures of 105° to 107° F., generalized rigidity and recurrent opisthotonic episodes accompanied by tachycardia and tachypnea. By the use of ice packs or special hypothermic apparatus the body temperature is reduced to a level of 90° to 95° F. In addition to these physical measures, the drugs of the "lytic cocktail" (chlorpromazine, promethazine, meperidine and levallorphan) (Table 2) are administered intravenously and intramuscularly to enhance the effect of the externally applied cold.

TABLE 1.—*Routine and Special Orders by Physician to Nurses Dealing with a Patient with Head Injury*

ROUTINE:

1. Blood pressure, pulse and respiration rate every 30 minutes until responding—then every two hours.
2. Rectal temperature every two hours.
3. Check ability to rouse, pupils and hand grips, every two hours.
4. Elevate head of bed 10 to 12 inches.
5. Put side rails on bed.
6. Administer oxygen constantly until responding. Keep airway clear with suction.
7. Nothing by mouth.
8. Sponge body with alcohol for temperature over 102° F.
9. Sedatives and analgesics.

SPECIAL:

1. Special nurses.
2. Hypothermia routine.
3. Tracheotomy routine.
4. Antibacterial therapy.
5. Intravenous isotonic and hypertonic fluids.
6. Blood transfusions.
7. Miscellaneous.

TABLE 2.—*Lytic Cocktail for Use in Treatment of Infants and Children with Head Injuries*

1. CHLORPROMAZINE HYDROCHLORIDE. (Thorazine)
   To reduce shivering.
2. PROMETHAZINE HYDROCHLORIDE. (Phenergan)
   Cutaneous vasodilator.
   Antihistaminic.
   Anticholinergic.
   Bronchial dilator.
3. MEPERIDINE HYDROCHLORIDE. (Demerol)
   Analgesic.
   Sedative.
   Spasmolytic.
   Cutaneous vasodilator.
4. LEVALLORPHAN TARTRATE. (Lorfan)
   Overcome drug-induced respiratory depression.

Evidence is now accumulating to indicate that these vigorous hypothermic methods are not only saving lives but restoring patients to useful positions in our society.

Electrolytes

Estimation of the serum electrolytes should be done frequently in all cases of severe head injury. Potassium levels tend to fall steadily in the presence of extensive cerebral injury. Hemoglobin values should be determined every four days, inasmuch as comatose and semicomatose persons tend to develop progressive anemia.

Activity

Ambulation is encouraged as soon as the condition of the child permits. If signs of fatigue become manifest, appropriate rest periods are instituted.

SKULL FRACTURE

Linear Fracture

Simple linear fractures of the vault require no surgical treatment. Basal fractures with rhinorrhea or otorrhea require prophylactic antibacterial therapy which is continued for at least three days after the cerebrospinal fluid fistula closes. Otorrhea rarely persists beyond the acute phase of the head injury, but rhinorrhea may become chronic and necessitate surgical intervention consisting of a reinforced closure of the dural laceration and repair of the defect in the floor of the skull with bone or one of the acceptable surgical metals or plastics.

Depressed Fracture

Small, shallow, depressed skull fractures may require no surgical treatment and will disappear with the growth of the skull. Larger depressed fractures should be elevated as soon as the condition of the child permits. "Ping-pong" fractures are easily managed by placing a marginal burr hole and pushing the bone outward with a blunt periosteal elevator. Comminuted, depressed fractures are managed by removing all the in-driven fragments.

Formerly, it was considered unwise to carry out a primary repair of the skull defect remaining after debridement of a compound depressed skull fracture. However, since the advent of antibacterial therapy replacement of the larger bone fragments has been carried out with excellent results from both the cosmetic and structural standpoints. If the wound is potentially infected, all of the bone fragments should be removed and the skull defect repaired later, using one of the accepted techniques for cranioplasty.

Brain Wounds

Brain wounds are treated by removing all devitalized tissue with suction. All in-driven bone fragments and foreign bodies are removed. Care is taken not to injure nervous tissue that is either functioning or may regain function. The dura overlying a brain wound is always closed in a water-tight manner. At times, this necessitates the use of a graft of temporalis fascia or periosteum.

Extradural and Acute Subdural Hematoma

Extradural and acute subdural hemorrhages constitute real surgical emergencies. The severe brain compression produced by these lesions may develop so rapidly that the patient dies before surgical measures can be begun. It must be remembered that in infants and young children extradural hemorrhage commonly occurs in the absence of skull fracture. The classical lucid interval may not occur. Furthermore, extradural hemorrhage may occur in the anterior and posterior fossae of the skull due to laceration of meningeal arteries or tearing of the venous sinuses.

Extradural and acute subdural hematomas are characterized by a more or less rapid deterioration of the state of consciousness, followed by pupillary inequality with dilatation of the ipsilateral pupil, slowing of pulse and respiration, increasing pulse pressure, rise of body temperature, progressive hemiparesis and, at times, generalized convulsions.

Acute Cerebral Edema

Generalized cerebral contusion with edema may produce a syndrome which is indistinguishable from that resulting from acute intracranial hemorrhage. Intracranial pressure may rise to critical levels. In such cases, standard trephine exploration in the frontal, temporal, parietal and suboccipital regions will disclose scattered areas of cerebral contusion without clot formation. Extensive bilateral subtemporal decompressions will often prove to be life-saving measures.

Chronic Subdural Hematoma

Chronic subdural hematoma is a comparatively late manifestation of head injury in infants and children. Intracranial hemorrhage of this type is due either to tearing of veins that extend from the surface of the brain to the major venous sinuses or to direct laceration of the venous sinuses. Venous injuries of this kind may occur in passage of the head through the birth canal, from maneuvers carried out to aid delivery or from a blow on the head.

In the acute phase the clot that forms is rather firm and bright red. With the passing of time it breaks down to form a more liquid, chocolate-brown mass. In the later phases it consists of clear xanthochromic fluid. Chronic subdural hematomas become surrounded by a membrane of varying thickness. These older hematomas will enlarge stead-

ily due to the osmotic attraction of the clot for cerebrospinal fluid.

The signs and symptoms of chronic subdural hematoma have no characteristic pattern. The lesion should be suspected in infants who have a bulging fontanelle, progressive head enlargement, listlessness, instability, poor appetite, vomiting, convulsions, hemiparesis and hyperactive deep tendon reflexes in varying combination. In older children, separation of the sutures, rather than a bulging fontanelle, may be the first sign.

Because these clots interfere with the growth of the brain, they should be recognized and treated as soon as possible. In the early stages, evacuation of the hematoma is accomplished by repeated needle aspiration of the subdural space through the lateral angle of the fontanelle. In the later stages, trephine exploration and drainage of the subdural space is the procedure of choice. If a well-developed membrane has formed, craniotomy is done and all solid clot and the membrane are excised.

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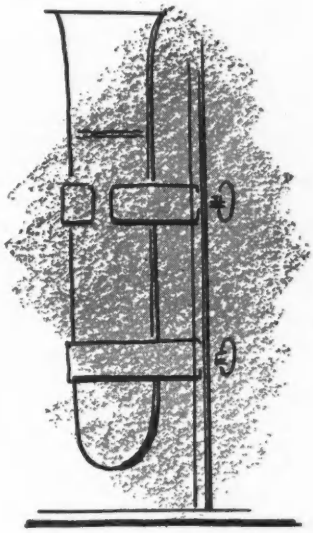

Trauma or Strain and Heart Disease

Causal Relationship

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• **Myocardial infarction and coronary insufficiency are the common conditions involved in workmen's compensation cases based on allegations of cardiac injury. Since coronary disease tends to be a progressive process, punctuated by sudden accidents, it is difficult to determine when a given stress at work may have had a significant effect in producing an infarction. Though there are gaps in our knowledge, considerable information is available indicating the way in which effort or stress may produce a myocardial infarction.**

Certain states have taken steps to develop more uniformity of medical opinion regarding causal relationship of stress to heart disease, and in other ways have attempted to improve the application of workmen's compensation laws in an attempt to provide fair and prompt compensation to those injured by their work but to discourage abuses of these laws.

"CAUSAL RELATIONSHIP of trauma to heart disease may exist under the following circumstances: Sudden death from acute coronary disease (or coronary occlusion with myocardial infarction, or acute coronary insufficiency) in which symptoms develop during the course of or immediately following exertion or strain that is both excessive and unusual for the particular individual concerned; this exertion or strain may be either physical or emotional."

The foregoing is a quotation from a guide for physicians regarding the relationship of trauma or strain to heart disease, developed by a special committee of the Washington State Heart Association. It was formulated in an attempt to clarify some of the problems of the cardiac under Workmen's Compensation laws.

This is a broad field which can be narrowed for purposes of this discussion, since we are dealing with coronary artery disease in most cases. That this is true is indicated by experience such as that of Texon,[14] who noted that 78 of 100 Workmen's Compensation claims based on heart damage were due to coronary artery disease. A review of claims in the ten years 1950-59 under industrial insurance law of the state of Washington showed that in 316 of 401 cases (77 per cent) in which a claim of cardiac damage was made, the problem was acute myocardial infarction or acute coronary insufficiency; and excessive or unusual effort was the alleged cause of the cardiac accident.

Although excessive effort may produce congestive failure if there is preexisting heart disease, this is an uncommon cause of cardiac claims. Industrially-related cerebral vascular accidents, secondary to hypertension, are also relatively uncommon. Although trauma to the chest, either penetrating or nonpenetrating, may injure the heart or cause death, cases of this kind make up a small portion of the total claims based on cardiac damage (less than 1 per cent of the Washington cases) and therefore will not be discussed here.

In light of the foregoing, the comments will be restricted to the effects of effort, stress or strain in precipitating cardiac accidents in persons with preexisting coronary atherosclerosis. Preexisting coronary disease has been specified, since it is generally agreed that even great stress or effort rarely harms the normal heart. It should be noted that the omission of cardiovascular diseases other than effort-related myocardial infarction from this discussion does not imply that in individual cases these other conditions are less important or that claims related to them less difficult to decide.

Almost everyone who has been interested in the Workmen's Compensation problems has been struck by the apparent disparity in the opinions of physicians, compensation administrators and courts as to the relationship of effort at work to heart disease. It is generally agreed that this variation in opinion stems from a lack of agreement among physicians as to when a given effort may have played a significant role in precipitating a heart attack or death. That there should be such disagreement is not strange, for the problem is a complicated one—the possible aggravating effect of a given stress on a progressive, degenerative disease which, in its natural course, is often punctuated by sudden worsening in the form of myocardial infarction or sudden death. What the factors are that precipitate coronary occlusion or sudden death is poorly understood. In most cases such attacks occur without a recognized significant event in the individual's activity or environment and, as a matter of fact, occur not uncommonly during sleep. A decision as to whether there

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is relation between a given effort at work and subsequent heart attack or death is made even more difficult by the observation of pathologists that a coronary occlusion may require hours or days to develop to the point of symptoms. The activities of the worker immediately before the onset of symptoms, therefore, may be unimportant in the process.

It is well to remember that there are two different problems involved—one related to the underlying atherosclerosis, the other to the thrombotic or occlusive complications of this process. With the rare exception of cor pulmonale, secondary to job-related pneumoconiosis, no occupational heart disease is recognized. However, there have been suggestions for some years that certain aspects of some jobs, particularly as they relate to emotional stress, may accelerate the development of atherosclerosis.[2] Proponents of this theory point out that stress may cause an elevation of serum cholesterol, which may accelerate the development of atherosclerosis. In addition, stress may cause an increase in pulse rate, cardiac output and blood pressure. Other as yet undefined ill effects of tension are also suspected. Friedman[2] and Russek[11] described a personality pattern common to young men with coronary disease, and emphasized the importance of occupational and other forms of stress in the development of coronary atherosclerosis. However, others are skeptical of the importance of emotional factors[6,13] and the question remains unanswered. Even assuming it may yet be proved that emotional stress hastens the process of atherosclerosis, it does not seem to deserve weighty consideration in a discussion of job-related heart disease, for these reasons: Atherosclerosis takes years to develop, and emotional tension is only one of many factors apparently affecting its development; there are many sources of stress in a given worker's life, some of which are totally unrelated to his job; it is difficult to quantify degrees of stress; and, as noted by Russek,[11] the individual's emotional reaction to his job is more important than his job classification, an observation supported by Pell and D'Alonzo,[9] who failed to find an increased prevalence of coronary disease in executive and managerial personnel, presumably subject to a greater responsibility and, therefore, stress.

It would appear that stresses and responsibilities that would be intolerable for one person are but a pleasant challenge to another. Although as physicians we consider emotional stress among other factors when advising patients regarding jobs, we are not justified in saying that any group of jobs, because of the emotional stress that may be involved, is harmful to people who have coronary heart disease. In addition, until we know much more of the causation of atherosclerosis, we must be very cautious in compensation cases about concluding that the continuing emotional stresses of a particular job over a period of years accelerated an atherosclerotic process and led to a heart attack or death sooner than it might otherwise have occurred.

In recent years, attention has been directed toward the effect of exercise on the atherosclerotic process. A widely quoted study is that of Morris and co-workers,[7] who compared the development of coronary disease in bus drivers with that in bus conductors, and in postmen with that in persons in sedentary jobs in the Postal Department of Great Britain. They believed their study proved that men in physically active jobs have a lower incidence of coronary heart disease in middle age than have men in physically inactive jobs, and also that the disease was not so severe in physically active workers. Other investigators who have done similar epidemiological studies have expressed the belief that additional factors, such as diet and emotions,[3,4] were more important determinants of atherosclerosis than exercise. However, as far as the topic of the moment is concerned, there is no evidence that moderate or even strenuous exercise over many years has a harmful effect on the course of atherosclerosis. Actually, the reverse seems to be true. It must be added that exercise of any degree may produce angina if the coronary artery is too narrowed to compensate for it.

The chief problem for discussion here is the relationship of physical or emotional stress to acute myocardial infarction. Information on this is available in two areas: (1) The physiological effects of stress upon atherosclerotic coronary arteries, and (2) epidemiological studies correlating attacks of myocardial infarction with antecedent physical activities and emotional stresses.

There are great deficiencies in knowledge of both areas. Admitting these deficiencies, let us consider the mechanisms of coronary occlusion and myocardial infarction in order to determine whether they may be influenced by effort or stress. Coronary occlusion in the majority of instances occurs in one of three ways, by progressive intimal thickening, as a phenomenon secondary to intimal hemorrhage and by intraluminal thrombosis. It is agreed that all these processes occur as a part of the disease process and usually with little relation to the patient's environment. The question is whether any of them may be produced or significantly influenced by environmental conditions.

Since reference has already been made to the lack of convincing evidence that atherosclerosis is a job-related disease, the matter of intimal thickening needs no further discussion.

Paterson[10] has been the leading exponent of the proposition that frequency and degree of intimal hemorrhage are important factors in coronary occlusion. He said it can be demonstrated that most intimal capillaries arise directly from the lumen of coronary arteries and not from vasa vasorum in the adventitia. This being true, he said, increases in systemic pressure are transmitted directly to such capillaries; therefore, increase in blood pressure secondary to physical exertion or emotional stress may rupture such capillaries, leading to significant thickening of the intima and in some cases to actual occlusion of the coronary artery. Even if occlusion does not occur, Paterson said, a thrombus may form on the overlying and altered endothelium. He noted such intimal hemorrhage at autopsy in 87 per cent of 58 cases of death from coronary occlusions and, on the basis of the foregoing reasoning, he expressed belief that coronary occlusion is often the result of physical exertion or emotional stress. Other investigators have not found so high an incidence of intimal hemorrhage and have expressed doubt it is as common a cause of coronary occlusion as Paterson believes. Yater and associates,[20] for example, found evidence of recent intimal hemorrhage in the coronary artery in only 6 per cent of 450 soldiers who died of acute coronary artery disease. Some other pathologists believe that intimal capillaries arise from vasa vasorum and are, therefore, not as susceptible to changes in systemic pressure as Paterson[10] suggested. Further, it is agreed that intimal hemorrhage often occurs as a part of the disease process without regard to stress or strain.

Although coronary thrombosis has occurred clinically in association with physical or emotional stress often enough to suggest a causal relationship, it has been difficult to explain the possible connection. At least three mechanisms have been proposed in addition to changes which may occur secondary to an intimal hemorrhage. One of the oldest such suggestions is rupture of an atheroma into the lumen of the coronary artery, leaving a rough spot on the intima on which a thrombus forms. The way in which effort causes the rupture has not been adequately explained although the factors described by Texon,[15] and referred to below, may be relevant. Evidence against this hypothesis is the lack of pathological proof that it occurs. Several observers have demonstrated an increased coagulability of the blood during acute emotional stress and have offered this as one of the causes of coronary thrombosis at such times.[2] Lastly, Texon[15] recently described the hemodynamic factors which not only influence the localization of atherosclerotic lesions but may even initiate the occlusive event. He referred to Bernoulli's theorem that fluid in motion possesses energy by virtue of its velocity and pressure. The static pressure plus the velocity pressure are constant as fluid flows in a tube of uniform size. However, according to Texon's concept, if there is narrowing at one point, the velocity increases and the static pressure falls correspondingly. The fall in static pressure at such a point produces a negative pressure or "suction" effect which stimulates intimal thickening and atherosclerotic plaque formation in this area. When degenerative changes in the plaque have progressed to a critical degree, an increase in velocity may increase the suction effect enough to detach the intimal layer and lead to thrombosis at this point. Such an increased velocity might occur with physical effort or emotional stress. Thus, we have four mechanisms which, theoretically, could lead to coronary thrombosis and occlusion. It should be kept in mind that even if stress initiated thrombus formation, it might take hours or days for narrowing to progress enough to cause symptoms.

The relation of coronary insufficiency to stress is easier to visualize. This is an extension of the process which produces angina pectoris but in which more prolonged disproportion between blood flow through the coronary arteries and blood needs of the myocardium leads to permanent, usually focal, myocardial necrosis. It occurs in the presence of preexisting coronary atherosclerosis which may have been unrecognized. It may, of course, be produced by anything that increases cardiac work, including physical effort or emotional stress, in addition to a number of conditions leading to decreased coronary flow, such as shock, hemorrhage and arrhythmia. It is generally agreed that this is a mechanism by which severe and prolonged effort or stress may lead to myocardial infarction even in the absence of coronary occlusion.

There are many statistical studies on the relationship of effort to myocardial infarction, among which the most widely quoted are those of Master,[5] purporting to show no relationship between effort and coronary occlusion, and those of French and Dock[1] and Yater and coworkers[20] suggesting there is relationship between unusual physical effort and myocardial infarction. Suffice it to say this is a subject which lends itself to bias and strong opinion, depending on the author's background and approach. The facts are few, the opinions many, divergent and often not entirely objective.

The American Heart Association, through its rehabilitation program, has been much interested in this problem, since fear of increased costs under industrial insurance acts has been one of the deterrents to employment of persons with heart disease. There are two committees of the American Heart Association working on the problem, the Trauma

and Strain Committee and the Medical, Legal and Insurance Committee.

The Trauma and Strain Committee has three projects under way:

1. The careful pathological examination of sudden deaths of heart disease in New York City correlated with the circumstances before death.[18]

2. A clinical study of the relation of events at work to heart attacks in a large New York industry.

3. A comprehensive investigation of legal procedures involved in decisions in compensation claims based on heart damage all over the United States.

Completion of the first two studies will shed more light on the possible relation of stress and strain to heart attack but cannot be expected to solve the problem.

Although the ultimate truths in this matter will probably elude us for some years, today's compensation cases based on allegation of heart damage cannot be postponed, and since the present methods of arriving at a decision in such cases are often unsatisfactory and unjust, some states have attempted to develop more logical and equitable procedures for handling compensation claims of this kind. Such attempts may be classified under four headings:

1. Development of medical criteria for the relationship of strain to heart disease;

2. Setting up of expert and unbiased panels to make such decisions;

3. Amendments to existing laws;

4. Improvement of application of existing laws.

Under Point 4 should be mentioned the improvement of rehabilitative efforts for cardiac claimants under Workmen's Compensation. There may well be other approaches.

In 1952, under the stimulus of the chairman of the Industrial Commission of the State of Utah, a group of internists in the Salt Lake area formulated criteria for the relation of strain to heart disease[16] which were accepted by their colleagues. In addition, they set up a system whereby each claim based on allegation of cardiac injury was examined by two or more experts from a panel chosen by the industrial commissioner from a list of specialists in that field that was submitted by the Utah State Medical Association.

After careful examination of the claimant by the panel, an opinion was submitted as to causal relation, this opinion being available before litigation occurred. According to Dr. L. E. Viko, one of the men chiefly responsible for the Utah plan and chairman of the cardiac panel, "Medical findings and Commission decisions are now reasonably consistent," and "knowledge of this has led to a marked decrease of the number of unreasonable claims." According to Industrial Commissioner O. A. Wiesley[19] of Utah, "Fear, suspicion, bias and hatred have been eliminated because expert, honest, medical opinion is available to both parties at no cost." Formal hearings by the Utah Industrial Commission have been decreased from an average of 300 a year to 40.

Similar criteria for the relation of strain to heart disease were formulated in the State of Washington in 1955 and a guide for physicians was published.[17] These criteria, a part of which has been quoted, have been approved by the three societies of internal medicine in the State of Washington and have been discussed in most of the county medical societies. The criteria are commonly referred to in heart disease compensation cases in Washington. The Department of Labor and Industries sends a copy of them with the file on each case related to heart disease to serve the examining physician as a guide in making his decision.

A panel of interested and qualified internists has been set up through the Washington State Heart Association and the Washington Society of Internal Medicine. Two or more of these panelists in different localities are selected by the Department to examine the living claimant or the file of the deceased. The panel decision has no legal status in Washington but, from our observations, appears to be usually accepted by the Department as the basis for its decision. It should be emphasized that the Heart Association became involved in the medico-legal problem through its rehabilitation program; that it is not for or against claimant or employer but is interested in a prompt and fair settlement of cardiac claims.

Three years ago, Oklahoma cardiologists drew up similar criteria[8] and distributed them to physicians in the state. Many states, of course, have procedures for using expert and impartial panels for review of accident claims in all fields of medicine.

Early in the Heart Association's investigation of the workmen's compensation situation in Washington, it was noted that some competent physicians were offering opinions as to causal relation that did not accord with accepted medical thought. When confronted with such opinions, their explanations often were based on their interpretation of the law (usually wrong) or their recognition of the financial needs of the claimant or survivors. Legal consultants have made it clear that as physicians we should make our judgments on purely medical grounds and should not warp our conclusions to thwart the law or to meet an economic need. This is a good rule to remember: We should practice medicine and let the courts interpret the law. If the

laws are inadequate or unjust, we should by other means attempt to influence a change through our legislators.

It would, of course, be desirable to know whether this combination of medical criteria and panel examinations does improve the application of the Workmen's Compensation Law in cardiac cases. If it is successful, it should lead to more uniform medical opinion regarding the effect of stress on the heart, to a reduction in the proportion of such cases proceeding to litigation, to prompter settlement of claims, to aggressive rehabilitation efforts and, lastly and most important, to better employment opportunities for persons with heart disease. Since there are many influences besides those of physicians that bear on the way workmen's compensation laws are administered, it is difficult to assess the relative importance of each. However, the Heart Association's committee has been encouraged by acceptance given to its efforts by physicians, administrators of the law, employers, labor unions and others. A study is under way in Washington reviewing cardiac claims since 1950 which will better evaluate the problem. One preliminary and interesting bit of evidence from this study is as follows: "There has been an almost steady decrease in the total number of accepted cardiac compensation cases, from 124 in 1950 to 68 in 1957."[12] This decrease stands in contrast to an increase in such cases reported in New York State and to frequent statements that such cases are increasing throughout the country. We who are working on the study were surprised to find this diminishing number of cases and, as yet, are unable to explain it satisfactorily.

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Chloride Content of the Cerebrospinal Fluid

H. W. GIERSON, M.D., and G. J. OWENS, M.D., Los Angeles

• **In a study of the spinal fluid chloride levels at the time of admission to hospital in a series of 1,788 cases of suspected meningitis, it was noted that the chloride content was not depressed in poliomyelitis, viral meningitis and encephalitis. In pyogenic meningitides, the spinal fluid chlorides were moderately depressed. More pronounced depression was noted in tuberculous and fungal meningitides.**

IN THEIR BASIC TEXT on the cerebrospinal fluid, Merritt and Fremont-Smith[3] frequently referred to the work of Mestrezat,[4] pointing out that he was the first to emphasize the diagnostic value of the spinal fluid chloride content. But whereas Mestrezat implied that the reduction in chloride content of the cerebrospinal fluid in pyogenic and tuberculous meningitis was part of the disease process, Merritt regarded the fall in chloride as a reflection of the decline in serum chloride. Mestrezat said that very low values were pathognomonic of tuberculous meningitis, but Merritt and Fremont-Smith noted that their results showed this not to be true, as their lowest values were in cases of acute purulent meningitis.

Parker[5] in his Textbook of Clinical Pathology (1948) said: "In acute meningitis, the chloride content is decreased usually below 600 mg. In tuberculous meningitis, the sodium chloride concentration is usually less than 650 mg. and often less than 550 mg. per 100 cc." And the following statement appears in Kolmer's Approved Laboratory Technic, 1951,[2] "Chloride values below 600 mg. are infrequent except in tuberculous meningitis, while values between 630 and 680 mg. per 100 cc. are commonly found in acute purulent meningitis." Still another viewpoint is implied in "A Syllabus of Laboratory Examinations in Clinical Diagnosis,"[1] the chloride content being omitted altogether from a table entitled "Cerebrospinal fluid in differential diagnosis."

Our clinical impressions resulting from the observation of more than 400 patients with tuberculous meningitis, plus lesser numbers with other meningitides, tended to support the statements of Mestrezat rather than those of his successors. This, plus the fact that our search of the literature failed to reveal any recent extensive clinical reports on the subject, prompted this study.

The 1,788 cases comprising this study were obtained from hospital laboratory records and were selected only if a spinal fluid chloride determination was made at the time of admission. The clinical records of the patients were then obtained, the diagnosis ascertained and the cases grouped by diagnosis as follows:

1. No central nervous system disease
2. Poliomyelitis
3. Meningitis, pyogenic
4. Meningitis, tuberculous
5. Meningitis, viral
6. Encephalitis
7. Organic central nervous system disease (tumors, cerebrovascular accidents, etc.)
8. Meningitis, fungal.

The cases in each of the foregoing categories were then divided according to the chloride content, per liter of spinal fluid:

A. 121 mEq. per L or higher
B. 117 through 120 mEq.
C. 113 through 116 mEq.
D. 108 through 112 mEq.
E. 100 through 107 mEq.
F. Below 100 mEq.

RESULTS

When the 1,788 cases were grouped according to diagnosis, the following distribution was obtained:

No central nervous system disease	507
Poliomyelitis	273
Meningitis, pyogenic	375
Meningitis, tuberculous	327
Meningitis, viral	125
Encephalitis	89
Organic central nervous system disease	78
Meningitis, fungal	14

The distribution by chloride levels for each of the above categories is shown in Table 1.

It will be noted in the table that the patterns of chloride content for poliomyelitis, viral meningitis and encephalitis are essentially similar to the one obtained in the group of cases in which no central nervous system disease was found. For the pyogenic meningitis group, a tendency toward lower levels is apparent. This tendency is even more pronounced in the tuberculous meningitis group.

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Presented before the Section on Internal Medicine at the 89th Annual Session of the California Medical Association, Los Angeles, February 21 to 24, 1960.

TABLE 1.—*Chloride Content of Spinal Fluid of Patients with Various Central Nervous System Diseases*

	Chloride Levels in mEq. per Liter					
	121 or higher	117 through 120	113 through 116	108 through 112	100 through 107	Below 100
No central nervous system disease	328	135	34	6	4	0
Poliomyelitis	202	58	10	2	1	0
Meningitis, pyogenic	129	112	64	43	20	7
Meningitis, tuberculous	48	38	49	58	78	56
Meningitis, viral	75	31	12	4	3	0
Encephalitis	57	24	6	1	0	1
Organic central nervous system disease	50	14	9	1	4	0
Meningitis, fungal	0	4	4	2	3	1

DISCUSSION

The significance of any single laboratory test result being subject to doubt, it is established policy at the Los Angeles County General Hospital to obtain serial spinal fluid specimens and to observe any tendency for the chloride level to rise or fall. But the severity of the illness in the various meningitides often denies the opportunity for such observation before the beginning of therapy, and some appraisal or interpretation of the initial findings must be made. It is precisely for this reason that only initial spinal fluid examinations were included in the study.

From the results obtained, it appears that the spinal fluid chlorides are unaltered in poliomyelitis, encephalitis and the viral meningitides. A moderate decrease in chloride content was associated with pyogenic meningitides, and a more decided decrease with tuberculous meningitis: In 70 of 375 cases of pyogenic meningitis (19 per cent), spinal fluid chloride content at admission was below 113 mEq. per liter, whereas it was below that level in 192 of 327 cases (60 per cent) of tuberculous meningitis. Furthermore, in only 7 per cent of the pyogenic meningitis cases was it below 108 mEq., against 41 per cent in the tuberculous meningitis group. In fungal meningitides, the spinal fluid chloride levels are quite similar to those in tuberculous meningitis.

Our experience indicated that the amount of chloride level in the spinal fluid is influenced by the amount in the circulating blood, but that the reduction noted in meningitis occurs primarily as a direct result of the disease process itself. It is, however, desirable to determine the amount in the spinal fluid and the amount in the blood simultaneously if a precise evaluation of the spinal fluid chloride level is to be made. The need for such simultaneous determinations is of special significance during the acute phase of the illness when electrolyte disturbances are more common.

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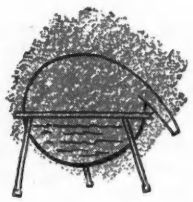

Cardiac Injury in Bodily Trauma

A Clinical Study

IRVING I. LASKY, *M.D.*, and JOHN H. DAVIS, *M.D.*, Beverly Hills

• **Involvement of the heart in bodily trauma occurs in a variable incidence. It is most frequently observed secondary to chest injuries. Because generalized bodily trauma can produce cardiac damage, the examination of the cardiovascular system is indicated and justified in cases of bodily injury.**

THE DEVELOPMENT of increasingly critical methods of clinical evaluation has facilitated the appraisal of the condition of the heart in cases of cardiac damage done by external impact. We now have serum glutamic-oxalacetic transaminase determination to augment the information supplied by history-taking, physical examination and the electrocardiogram.

Glutamic-oxalacetic transaminase is widely distributed in animal tissues, with the greatest concentration in heart muscle, skeletal muscle, brain, liver and kidney, in that order.[1] Injury to these organs increases circulating transaminase.

ANATOMICAL CONSIDERATIONS

The anatomic location of the heart makes it susceptible to injury as the result of accelerating, decelerating or crushing forces.

The heart lies in the middle mediastinum. Laterally, it is well protected by adjacent pulmonary structures. Anteriorly and posteriorly, however, the contiguity of this organ to the sternum and thoracic vertebrae exposes it to relatively immobile points of contact. Superiorly, very stout ligaments secure the base. The apex, on the other hand, is movable, the pericardial sac limiting the extent of motion.

Although anatomical protection is afforded, the relationship of the heart to boney structures of the thorax makes it vulnerable to injury of certain types, much as the brain can be injured by trauma to the skull.

PATHOGENESIS

Cardiac injury in bodily trauma may occur in any of five ways.[3]

1. A direct blow to the heart, such as might occur with fracture of the sternum or ribs.
2. Contusion or compression of the heart between the sternum and the vertebral bodies.
3. Application of indirect forces, as by compression of the legs and abdomen, producing increase of intraventricular pressure and subsequent myocardial damage.
4. Laceration of the abdominal or thoracic viscera, producing sudden increase in intraventricular pressure and subsequent myocardial damage.
5. "Concussion of the heart" (vagotonia) as manifested by arrhythmias.

Generalized blunt force, severe enough to crush or compress the chest and produce cardiac and other intrathoracic injury, is most often associated with vehicle accidents and falls from a height.

PATHOLOGY

A crushing downward force applied anteriorly may dislodge the heart from its superior and posterior attachments and tear the pericardial sac. Injuries of this magnitude are most frequently associated with severe chest and bodily involvement and are usually fatal. Less violence may cause incomplete internal transverse lacerations of one or both auricles above the auriculoventricular valve rings, and lacerations of attachments of the venae cavae and pulmonic veins may occur. Mural thrombi may develop over the site of injury in the auricle if the injured person survives. Crushing of the chest may produce punctures and lacerations of the pericardium and heart from sharp ends of broken ribs or the sternum. Sudden violent compression of the chest may cause one or more ruptures of the heart involving the ventricles, auricles or septa. In such cases death ensues rapidly from intrapericardial hemorrhage.

A severe localized blunt force, like that from the impact of a small, hard, fast-moving object striking the anterior chest wall, may contuse and rupture the thin anterior wall of the right ventricle. The great majority of isolated contusions of the heart occurring in the anterior wall of the right ventricle are caused by localized impact of that kind.[4]

At autopsy of 31 patients who died within three weeks of slight to moderate chest trauma but with severe extracardiac injury, 21 were found to have

Submitted August 3, 1960.

From the School of Medicine, University of California, Los Angeles 24.

TABLE 1.—*Follow-Up Studies of Patients with Cardiac Damage Incurred in Bodily Trauma*

Case	Age (Years)	Date	Cause	Evidence of Cardiac Injury	Transaminase Peak (SGO Units)	Reexamination
1.	24	7-11-55	Fell from motorcycle. Unconscious. Multiple abrasions of face and laceration of forehead.	T waves inverted Leads III, aVF. Flattened V_5, V_6.	78 (19 hrs.)	6-25-57–No cardiovascular findings. Transaminase normal. EKG normal except for infrequent ventricular premature contractions.
2.	31	7-17-55	Auto vs. auto. Driving. Thrown from car. Fracture 12th left rib.	Flattened T waves Leads III, aVF, V_5, V_6. Spiked T in V_2. Improved in 5 days.	232 (6 hrs.)	8-1-57–No cardiovascular findings. EKG and transaminase normal.
3.	30	7-21-55	Fell from second story roof. Unconscious. Fracture of pelvis, hands, L-5 vertebra. Lacerated bladder.	Transient T wave and ST segment changes in posterolateral leads.	211 (37½ hrs.)	9-17-57–No cardiovascular findings. EKG and transaminase normal.
4.	59	8- 9-55	Auto vs. auto. Driving. Fracture left 5th-8th ribs. Patient in shock.	Left ventricular hypertrophy with strain pattern. Possible cardiac dilatation. Changes persisted after recovery from shock.	78 (108 hrs.)	7-5-57 – No cardiovascular complaints. B.P. 170/110. EKG and transaminase normal.
5.	23	4-23-56	Motorcycle accident. Fracture — dislocation left ankle. Fracture left clavicle.	Subjective dyspnea, mild. Transient systolic murmur. Inverted T waves in Leads III, aVF. Occasional premature ventricular beats.	109 (2 hrs.)	9-11-57–No cardiovascular complaints. EKG and transaminase normal.

subpericardial hemorrhage, and small pericardial or epicardial tears were observed in ten. Subpericardial fibrosis was frequent among a similar group of patients who lived six to 18 months after the accident.[5]

CLINICAL EVALUATION

The most important symptoms, physical signs and electrocardiographic changes that occur in cardiac injuries are as follows:

Symptoms: Precordial pain; dyspnea and orthopnea; cyanosis; evidence of shock (gray pallor, weakness, sweating and restlessness).

Physical Signs: Cardiac arrhythmia; fluctuations in blood pressure; increase in the area of cardiac dullness (suggesting pericardial effusion or cardiac dilatation); paradoxical pulse; abnormal heart sounds; pericardial friction rub; cardiac murmurs if they appear suddenly or change abruptly; signs of cardiac failure, such as pulmonary edema or right heart failure.

Electrocardiographic Abnormalities: Extrasystoles; auricular fibrillation; auricular flutter and other disorders of rhythm; abnormal P waves; change in the QRS complex; abnormalities in the ST segment and T wave.

For a diagnosis of cardiac injury, Lowry recommended there be definite electrocardiographic evidence of myocardial damage within 48 hours after injury. The electrocardiographic changes in Lowry's series included variability in the ST segment, T wave changes, first degree heart block, transient bigeminal rhythm and ventricular extrasystoles.[7]

Lieberman and coworkers reported a series of 51 persons of all ages involved in various accidents,[6] nine of whom had evidence of cardiac injury. The evidence consisting of elevated serum transaminase and electrocardiographic abnormalities in all cases. The electrocardiographic changes involved combinations of T wave flattening and inversion, ST segment shifts, transient spiking of the T waves over the precordium and premature auricular and ventricular beats. Four of the nine patients had been injured while driving an automobile and three while riding a motorcycle, one in falling from a moving car and one in a fall from a second story roof. All patients had multiple contusions and eight had fractures. Five of the nine patients were carefully reexamined after varying periods from the time of the injury and the observations are shown in Table 1.

The following report illustrates many of the features of cardiac injury from external impact.

REPORT OF A CASE

A registered nurse, aged 52, was involved in a collision of automobiles September 28, 1959 at 7:45 a.m. She was thrown forward and struck her chest and face against the steering wheel. She was momentarily unconscious. She complained continually of pain in the chest and in the right cheek. The patient

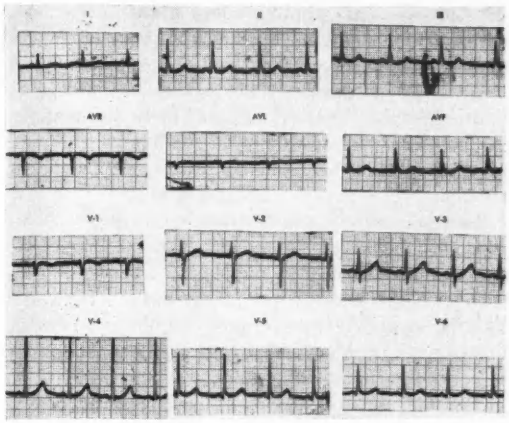

Figure 1.—Electrocardiogram taken five and a half hours after injury.

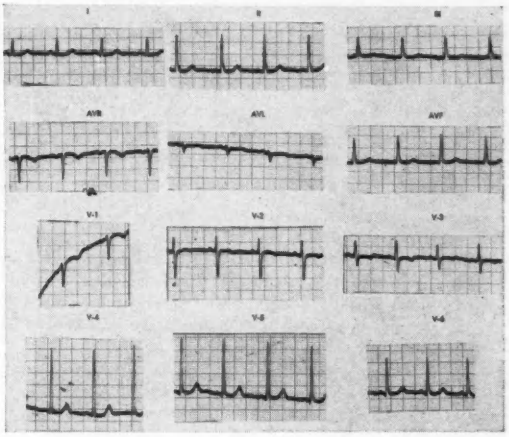

Figure 2.—Electrocardiogram taken 24 hours after injury.

was examined at 12:20 p.m. The blood pressure was 120/80 mm. of mercury and the pulse was regular at a rate of 78. The only abnormality noted on physical examination was a depression over the right malar bone and some ecchymosis at the site. However, the patient complained of persistent pain over the chest, not entirely relieved by narcotics. At 1:30 p.m. an electrocardiogram was ordered because of suspicion of injury to the myocardium (see Figure 1) but the tracing was completely within normal limits. The pain became progressively worse. Serum transaminase was 370 SGO-T units. The blood sedimentation rate was 28 mm. in one hour. Leukocytes numbered 12,500 per cu. mm., with 93 per cent polymorphonuclear cells. An electrocardiogram on the morning of September 29 showed notching and widening of the P waves in Leads II, III and aVL, flattening of the T in Leads III and diphasic T waves in V_2 and V_3 (see Figure 2). Serum transaminase on this date was 230 SGO-T units. On September 30 an electrocardiogram showed slight depression of the ST segments in Leads III and aVF, a flat T wave in aVL and a tendency of the T waves to become upright in V_2 and V_3 (see Figure 3). On this date the patient complained of severe pain in the chest, and a to-and-fro friction rub was heard below the sternal xyphoid junction. An x-ray film of the chest was within normal limits. On October 1 the pain was still present but the friction rub had disappeared and by the following day the pain was considerably diminished. Microscopic hematuria developed and a urologist attributed it to renal contusion. An electrocardiogram on October 2 showed diphasic T waves in V_2 and V_3 and depression of the ST segments from V_2 to V_5. On October 3 the serum transaminase was 47 SGO-T units. By October 6 the chest pains had practically disappeared. On October 7 the electrocardiogram had

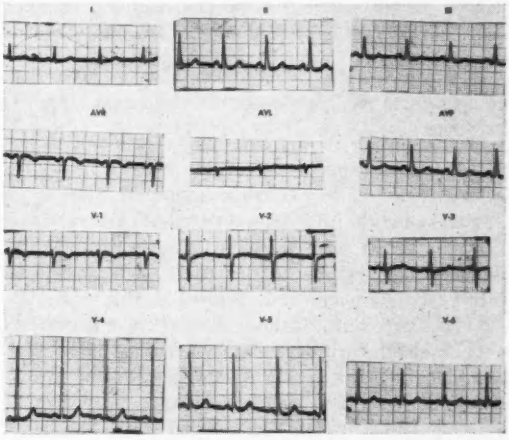

Figure 3.—Electrocardiogram taken 48 hours after injury.

practically returned to normal except for minimal depression of the ST segments in V_2, V_3 and V_4. On October 8 the serum transaminase was 12 SGO-T units. The next day the electrocardiogram again showed T wave inversion in V_2 and a lowering of the T wave in V_3 and slight ST depression in V_3. On October 13 the T wave was less deeply inverted in V_2. On October 19 the electrocardiogram was entirely normal except that the T wave remained inverted in V_2 (See Figure 4).

On October 29 the chest pains had entirely disappeared and vital signs were within normal limits. The malar fracture was repaired and, at a later date, a hysterectomy for a fibromyomata was performed, all without incident. On October 30 a vectocardiogram was reported as abnormal, showing a superior and slightly anterior ST displacement without evidence of loss of ventricular potential. On March 24, 1960 the electrocardiogram was within normal limits

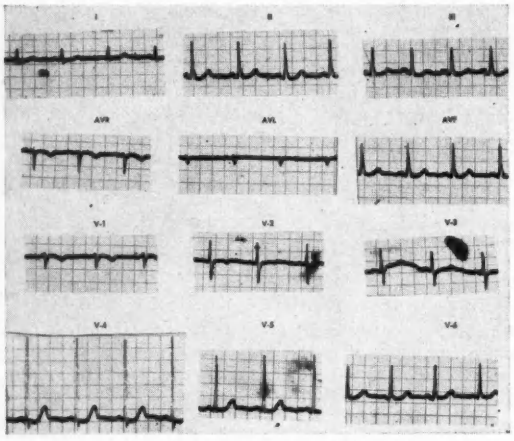

Figure 4.—Electrocardiogram taken 20 days after injury.

except for an inverted T in aVL and V_2, changes that had not been present on the original electrocardiogram.

DISCUSSION

Barber[2] reported the first clinical attempt to determine the incidence of cardiac trauma in bodily injuries. He made electrocardiographic studies in 33 cases of chest injuries and noted abnormalities in eight of them (24.2 per cent). Sigler studied the incidence of trauma to the heart in 42 cases of rather serious accidental injuries to the body.[8] In 32 (76.2 per cent) of these cases there was clinical and/or electrocardiographic evidence of some cardiac damage. One patient died of the cardiac injury and one showed permanent damage, but the remainder had complete recovery so far as the heart was concerned. Of 75 patients studied by Lowry, 20 (26 per cent) showed some slight but definite cardiac abnormalities within 48 hours after the accident.[7]

Although in general there is cardiac damage in approximately one-third of cases of bodily trauma, the incidence varies widely, depending on the kind of case studied and the location and extent of trauma.

As demonstrated in the extensive work of Lieberman and co-workers,[6] the serum glutamic-oxalacetic transaminase levels may be elevated in cases of generalized bodily trauma without cardiac involvement.[6] This is, of course, confirmatory of the presence of this enzyme in other major organs of the body and, in particular, in skeletal muscles.[1]

The literature contains few examples in which there is evidence of continued cardiac disorder following recovery from injury. Death or complete recovery is the ordinary expectation.

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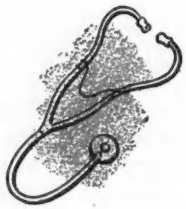

Iatrogenic Perforation of the Esophagus

JOHN H. HEALD, *M.D.*, San Francisco

• **In three of ten cases of instrumental esophageal perforation occurring at the San Francisco Hospital in the period 1947-1959, the patient died, having received supportive therapy only. Eight of the ten perforations in this series resulted from the use of the esophagoscope.**

Roentgen studies of the neck and chest, including studies with an opaque medium in the esophagus, are invaluable aids in establishing the diagnosis.

Close observation of all patients for twenty-four hours after instrumental procedures involving the esophagus is advisable.

THE LITERATURE contains many detailed reports on esophageal perforation, mainly describing the clinical and therapeutic aspects of spontaneous rupture. The purpose of this report is to emphasize the hazards of instrumental procedures involving the esophagus.

During the period 1947-1957, there were ten instances of instrumental esophageal perforation at the San Francisco Hospital. Review of the records since that time revealed no additional accidental ruptures through October 1959. The procedures preceding perforation, the diagnostic measures available to confirm the diagnosis and the therapeutic outcome will be briefly discussed. The age range of patients was from 18 months to 75 years. Of the ten instrumental perforations, eight resulted from use of an esophagoscope. Most of the perforations occurred at site of previous esophageal disease. In three cases the site was the cervical esophagus at the level of the cricopharyngeus muscle.

DIAGNOSIS

The clinical diagnosis is based on some or all of the following symptoms: Pain, substernal, frequently radiating to the back, cervical or upper abdominal; dyspnea, dysphagia and vomiting. Many observers have noted that morphine does not relieve the pain. Physical findings include shock, occasional cyanosis, elevated temperature, swelling of the soft tissues in the lower neck, effusion or consolidation and rigidity of the upper abdomen. Subcutaneous emphysema or the presence of a "mediastinal crunch" reportedly occurs in approximately fifty per cent of the cases. The symptoms vary depending on the site of perforation but, in general, rupture of the thoracic esophagus is more dramatic and fulminating in onset than is rupture in the cervical esophagus. Perforation in the lower third is not infrequently accompanied with upper abdominal pain and rigidity.

Instrumental esophageal perforations are ordinarily not recognized at the time of rupture, but in each of the ten cases at this hospital, the patient complained of rather severe cervical or thoracic pain soon after the procedure, the body temperature rose promptly and dyspnea developed. The most important point in establishing the diagnosis is that the clinician in charge of the patient after an instrumental procedure be aware of the possibility of perforation.

According to Gay,[2] left hydropneumothorax is present in 50 per cent of cases of spontaneous perforation of the esophagus, bilateral in 25 per cent, and on the right in about 7 per cent. Cervical or mediastinal emphysema is present in over 50 per cent. Some observers have made a point of the absence of free peritoneal or retroperitoneal air in such cases; and while this was true in the present series also, there has been at least one case of spontaneous rupture of the esophagus at this hospital with demonstrable retroperitoneal air. In addition to the roentgen observations on plain films, opaque studies of the esophagus using media described below should be done for localization of the site of perforation. Even if the site of perforation cannot be shown by such studies, this fact does not rule out the possibility of rupture, and they are and should always be carried out promptly if the condition of the patient permits. Most clinicians oppose the use of barium and advocate opaque oil such as Lipiodol® or similar iodine preparations. However, it has not been the experience at this hospital that barium *per se* in the mediastinum, in the pleural or peritoneal cavity, has caused tissue injury. In two of the present ten cases, barium was used to outline the site of perforation and caused no complication. Nevertheless, when apprised of the possibility of esophageal rupture, it is our policy to use opaque media other than barium. If the clinician is aware of the

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Presented before the Section on Radiology at the 89th Annual Session of the California Medical Association, Los Angeles, February 21 to 24, 1960.

TABLE 1.—*Clinical Data in Ten Cases of Iatrogenic Perforation of the Esophagus*

Case No.	Age	Sex	Diagnosis and Procedure	Perforation Location	Roentgen Findings	Therapy	Result
1.	75	M	Carcinoma of esophagus Esophagoscopy	Middle third	Right lower lobe consolidation. Gastric tube in right lower quad.	Supportive	Died
2.	38	M	Esophageal varices Sengstaken tube	Lower third	Perforation shown with Lipiodol.® Tube in rt. pleural space.	Primary closure and drainage	Died
3.	1½	F	Lye stricture mid-third Esophagoscopy and bougie	Middle third	Mediastinal and subcutaneous emphysema. Effusion — pericardial and pleural.	Supportive	Died
4.	60	F	Hiatal hernia esophagitis Esophagoscopy	Junction upper and middle third	Perforation shown with barium.	Supportive	Excellent
5.	63	F	Hiatal hernia Esophagoscopy	Lower third	Left hydropneumothorax.	Drainage	Excellent
6.	51	F	Suspected foreign body Esophagoscopy	Upper third	Subcutaneous emphysema.	Drainage	Excellent
7.	74	M	Hiatal hernia Esophagoscopy	Junction upper and middle third	Subcutaneous emphysema.	Drainage	Good (divertic. at perf. site)
8.	69	F	Carcinoma of esophagus Esophagoscopy	Upper third	Subcutaneous emphysema. Perf. shown with Lipiodol.	Drainage	Excellent
9.	53	M	Anoxia Tracheotomy (Broken B-P blade)	Upper third	Paraesophageal abscess. Foreign body in esophagus.	Primary closure and drainage	Excellent
10.	47	F	Chemical burn Esophagoscopy	Lower third	Right empyema perf. shown with barium.	Drainage decortication	Excellent

possibility of perforation and roentgen study is done promptly, the diagnosis and site of perforation should be established within a few hours of rupture.

THERAPY

The detailed management of a patient with instrumental esophageal perforation is beyond the scope of this presentation but a few observations pertinent to the management of the ten patients in the present series are worth discussion. In general, aggressive surgical management provided the most satisfactory result. But whether the procedure involved drainage of the area or primary closure of the esophagus with placement of drains, did not influence the outcome significantly in this series. Of the three patients receiving only supportive management, two died. In one case (that of the only other patient in this series who died) primary closure with drainage was not done. However, as can be seen in Figure 2, the perforation in that case was most unusual; the rent was more than 12 cm. long. In one of the seven surviving patients a diverticulum developed at the site of perforation. In this small series there was no valid correlation between the promptness of operation after perforation occurred and the therapeutic outcome.

REPORTS OF CASES

The following four cases are reported to illustrate some of the complications of esophageal perforation.

CASE 1. A 75-year-old man entered the hospital complaining of coughing and progressive difficulty in swallowing of five days' duration. Although he had noted some difficulty in swallowing solid foods for approximately a year, it was only during the week before admission that swallowing any food caused coughing. He had lost approximately ten pounds in weight. The patient appeared to be chronically ill but in no acute distress. Blood pressure was 160/82 mm. of mercury, pulse rate 120, respiration 24 per minute, and body temperature 102° F. There was a Grade II blowing apical systolic murmur. The heart was otherwise not remarkable. The lungs were clear. The clinical impression was carcinoma of the esophagus with tracheoesophageal fistula.

Following roentgen studies of the esophagus, an esophagoscope was introduced and the stricture was dilated. Esophagoscope examination was done five days later, at which time a biopsy specimen of the esophagus was taken and a polyethylene tube was thought to be inserted through the stenosis of the midesophagus into the stomach. Twenty-four hours

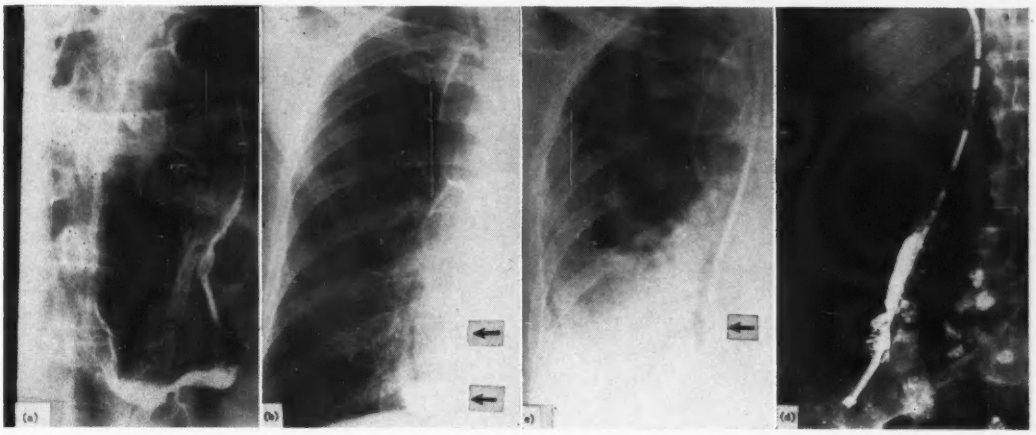

Figure 1.—Esophageal perforation due to a combination of carcinoma, esophagoscopy and intubation. (a) Spot film showing pronounced dilatation of esophagus with almost complete stenosis at level of bifurcation. (b) Conventional chest examination showing the dilated esophagus, barium in bronchi of the right lower lobe (*arrow*), and mediastinal abscess (*arrows*) (*retouched*). (c) Follow-up study of chest after the esophagoscopy demonstrated right lower lobe consolidation and course of tube through mediastinal abscess (*arrow*). (d) Gastric tube lying freely in peritoneal cavity with tip in right lower quadrant; tube presumably perforated the inferior border of the abscess and the diaphragm.

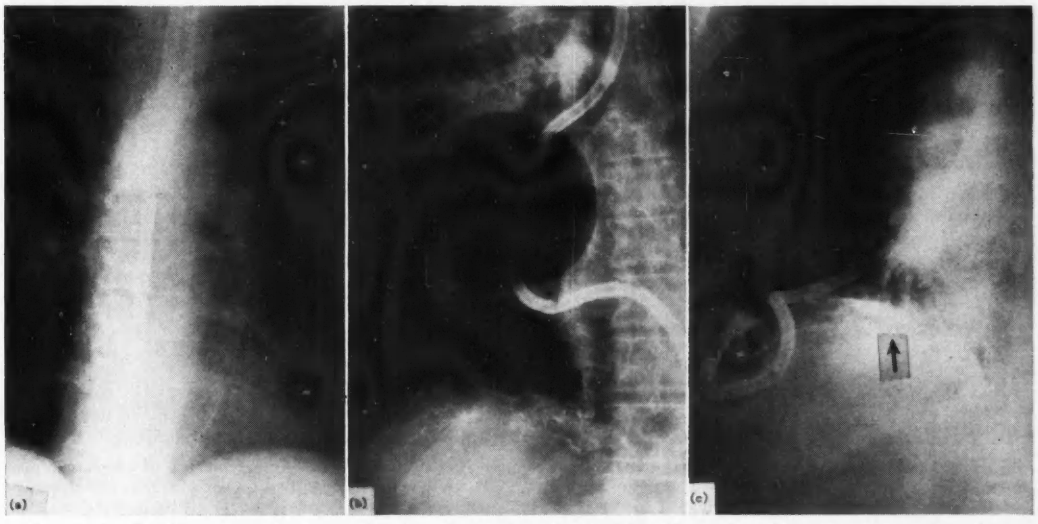

Figure 2.—Esophageal perforation following intubation. (a) Double-balloon tube (Sengstaken) in midesophagus; prior to taking the film, the attending physician felt the balloon was in the gastric fundus. (b) Tube following withdrawal, re-insertion and re-inflation. A film was not obtained prior to re-inflation, but this film demonstrates a fully inflated balloon free in the right pleural space adjacent to the right midmediastinum. (c) Balloon deflated. Further travel of the tube into the right pleural space. Note Lipiodol® between lung and diaphragm (*arrow*).

later the patient was noted to be dyspneic and there were moist rales throughout both lower lung fields. Thirty hours later there was an area of dullness at the right base and roentgen examination revealed consolidation in the right lower lung field. A small amount of Lipiodol® was injected into the polyethylene tube and roentgen studies were carried out. (Figure 1). No operative procedure was attempted. The patient received supportive therapy only and died the following morning.

Case 2. A 38-year-old man, a chronic alcoholic, entered the hospital with complaint of vomiting blood of three days' duration. He had been in the hospital a year previously with a similar complaint and was discharged with a diagnosis of Laennec's cirrhosis and esophageal varices. A month before the present admission the patient ceased eating and drank with increasing frequency. During this time any attempt at swallowing solid foods resulted in vomiting and he had repeated dark stools. Upon

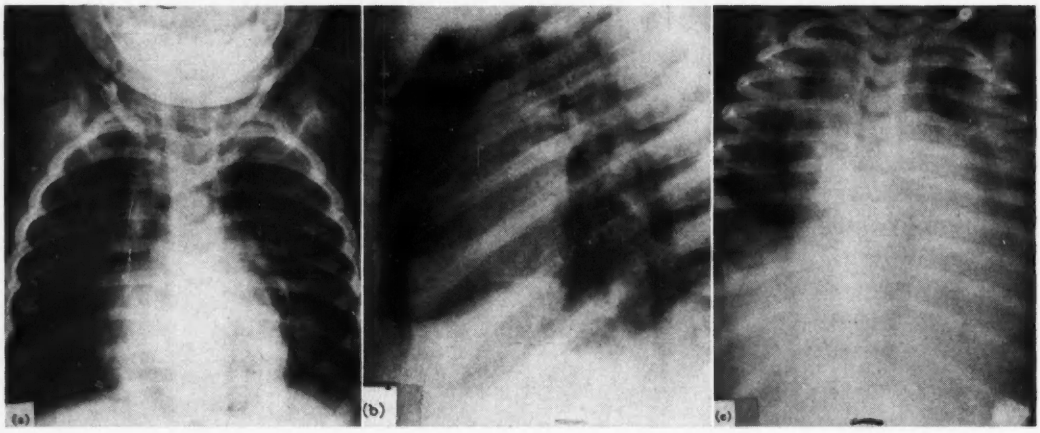

Figure 3.—Esophageal perforation following esophagoscopy and bouginage. (a), (b) Mediastinal and subcutaneous emphysema following esophagoscope perforation. (c) Infected pleural and pericardial fluid subsequent to perforation. Film made four days after a and b: patient expired two days later.

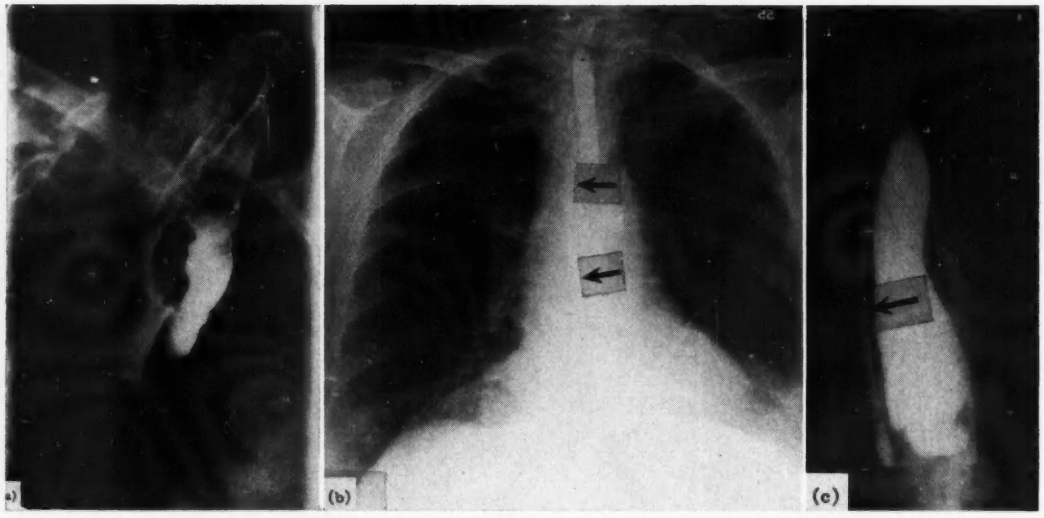

Figure 4.—Esophageal perforation following esophagoscopy. (a) Spot film showing perforation of esophagus near junction of upper and middle third outlined by barium. (b) Mediastinal tract opacified by barium; the esophagoscope had traversed this area. (c) Persistence of mediastinal tract outlined by barium one year following perforation.

physical examination he was observed to be poorly developed, undernourished, icteric and semiconscious. Blood pressure was 130/80 mm. of mercury, the pulse rate 84, and body temperature 99.2° F. Multiple spider angiomata were present over the arms and shoulders. The lungs were clear and the heart sounds were within normal limits. The liver was greatly enlarged.

A Sengstaken tube was passed, the stomach lavaged and an antacid drip was commenced. Roentgen examination at this time (Figure 2) showed the tube to be in the esophagus. It was withdrawn, reinserted, and filled with air. There was immediate pain in the right upper quadrant, radiating to the back, the pulse rate increased to 130, the temperature rose to 102° F. and mediastinal crepitus was present. Approximately four hours from the time of rupture, the patient was taken to surgery. A right thoracotomy incision revealed 1,000 cc. of blood and gastric contents in the right pleural space. The Sengstaken tube was lying free in the right pleural space and there were early inflammatory changes on all pleural surfaces. There was a rent in the esophagus 12 cm. in length, commencing at the junction of the middle and distal thirds. The tube was inserted through the distal intact esophagus, the rent was closed with interrupted sutures, and drains were placed in the right pleural space. Transfusions of

thirteen units of blood were given but the patient died the following morning. At autopsy, multiple esophageal varices in addition to Laennec's cirrhosis were noted.

CASE 3. An 18-month-old girl entered the hospital for dilation of a stricture of the esophagus. Approximately fifteen months previously she had been admitted to the hospital following lye ingestion and a stricture of the midesophagus was subsequently demonstrated. Barium studies of the esophagus showed an area of stenosis at the level of the bifurcation of the trachea. Distal to the stenotic area the lumen was normal.

An esophagoscope was introduced and the esophagus was dilated with bougies at the point of stricture. Several hours later fullness was noted in both sides of the neck and roentgen examination (Figure 3) revealed subcutaneous and mediastinal emphysema. Large doses of antibiotics and supportive therapy were prescribed. There was a steady decline in the condition of the patient and she died six days later. At autopsy a 1 cm. perforation at the site of stenosis was observed.

CASE 4. A 60-year-old woman entered the hospital with complaint of difficulty in swallowing of several months' duration. The food seemed to stick in the lower end of the chest and she had occasional epigastric distress after eating. Upon esophagoscopic examination, a hiatal hernia was observed, with evidence of esophagitis. The hernia was subsequently repaired. When examined again several months later the patient still had difficulty in swallowing. Hiatal hernia again was observed. Twice more esophagoscopic examination was carried out, and the second time considerable inflammatory changes were seen in the distal esophagus. As there was also a moderate amount of bleeding, the procedure was discontinued. Two hours later the patient had severe pain in the chest and roentgen study with a barium swallow revealed extravasation of the material from the esophagus at the level of the thoracic inlet (Figure 4). Large doses of antibiotics and supportive fluids were prescribed and the patient recovered.

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Fatal Epistaxis

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• **The instances in which nosebleed is potentially fatal are those in which there is a history of recent head injury, severe arteriosclerotic cardiovascular disease or an underlying vascular tumor in the nasal chambers. Fatal nasal bleeding has not been reported in children. An awareness on the part of the physician of the potentially fatal significance of his patient's nosebleed is the very best insurance against such an event. Intelligent history-taking, careful physical and x-ray examination, generous sedation, precise local cauterization and packing, estimation of hemoglobin mass and a search for bleeding and clotting disorders are the best weapons of the physician called to treat epistaxis. These procedures, coupled with adequate blood replacement and an informed attitude toward surgical interruption of the blood supply to the bleeding region should forestall death from fatal nosebleed.**

ON THE NIGHT of November 27, 1957, a 27-year-old Negro man was admitted to Wadsworth General Hospital, Veterans Administration Center, Los Angeles, with a diagnosis of anemia of undetermined origin. Late that night, the genesis of the anemia became clear when a massive nasal hemorrhage developed that responded only to the insertion of a large postnasal pack. Questioning of the patient and his relatives elicited that three months before he had been in hospital elsewhere following a motorcycle accident in which he received a closed head injury that resulted in left hemiparesis and recurrent nosebleeds. The patient said that he had not had nosebleeds before the accident and that the episodes of bleeding were not preceded by nasal manipulation or physical exertion. There was no history of sickle cell disease, easy bruising, jaundice or familial bleeding tendencies.

The patient was pale and his memory poor. Also noted were left peripheral facial nerve paralysis, diminished sensation over the distribution of the left trigeminal nerve and a conductive hearing loss in the left ear. The hematocrit was 24 per cent, the hemoglobin content was 7.2 gm. per 100 cc. of blood, leukocytes numbered 5,050 per cu. mm. with a cell differential within normal limits, and bleeding and clotting times were normal. A Rumpel-Leede test was negative for capillary fragility. Careful review of the roentgenograms of the skull and face showed only a fracture of the left zygoma.

During his stay in the hospital the patient had several episodes of sudden, spontaneous, massive bleeding from the nose and mouth, controlled only by insertion of large postnasal packs and necessitating several transfusions. Because of the persistence and severity of these nosebleeds, the left external carotid artery was ligated on the morning of December 14, 1957. Nasal packing was removed and the patient apparently did well following this operation until, at 6 p.m., cataclysmic hemorrhage from the nasopharynx developed suddenly. Within three minutes of the onset of this hemorrhage a postnasal pack and anterior nasal pack were reinserted and a venous cutdown was begun. In five minutes, however, the patient was dead.

At autopsy a rent was noted in the intracranial portion of the left internal carotid artery which communicated with the interior of the sphenoid sinus and through the sphenoid ostium with the nasopharynx. Death might possibly have been averted had carotid angiography been included in the diagnostic studies which were undertaken to find the cause of epistaxis.

CASE 2. On December 30, 1957, a 68-year-old white man entered Wadsworth General Hospital for treatment of severe left-sided epistaxis. In January of 1956 he had been in hospital for the same disorder and, at that time, was bleeding from a point below the posterior tip of the left inferior turbinate. In April of 1957, he had been brought into the hospital for treatment of arteriosclerotic heart disease with angina pectoris. A recent examination directed at a disturbance of bowel function had indicated the presence of an annular lesion of the large bowel. On December 29, 1957, bleeding again commenced from the left side of the nose and the patient was brought into the hospital on the following day. There was no other history of bleeding diathesis.

Upon physical examination he was observed to be pale, calm and fairly alert, with definite slurring of speech but no obvious motor weakness. He complained of continuous pain in the chest and in both

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arms. The blood pressure was 150/90 mm. of mercury. The pulse rate and temperature were within normal limits. Upon removal of a nasal pack that had been placed to stop the bleeding, epistaxis did not recur. No evidence was seen of tumor or infection of the nasal chambers or paranasal sinuses. The heart was not enlarged and there were no murmurs or rubs. The lungs were clear. Hemoglobin was 9.8 gm. per 100 cc. of blood and the hematocrit was 29 per cent. Leukocytes numbered 5,500 per cu. mm. A roentgenogram of the chest showed the heart, great vessels and lung fields to be within normal limits. An electrocardiogram was interpreted as consistent with subendocardial injury.

The patient was treated with nitroglycerin, ferrous sulfate, sedatives and bed rest. Bleeding from the nose did not recur, but the patient continued to complain of discomfort in the chest and arms. Because of the anemia and of coronary insufficiency indicated by progressive electrocardiographic changes, a transfusion was begun at 6:30 p.m. on January 7, 1958. Within a half hour the patient began to perspire profusely and the skin became ashen. He complained of fairly severe pain in the chest. At 8:15 p.m. respirations became labored and oxygen was administered. At 8:30 p.m. the patient died.

Permission to perform examination postmortem could not be obtained. While the nosebleed in that case may appear to have been incidental to the already existing coronary artery disease and bowel carcinoma, the addition of blood loss may have upset the tenuous cardiovascular equilibrium enough to bring about intolerable coronary insufficiency. The possibility that death was caused by a reaction to the transfusion was considered, but was discarded after consultation with the medical staff and examination of the blood remaining in the transfusion flask for mismatching and bacterial contamination.

The feelings of profound chagrin and defeat arising from my personal experience in treating these two patients led me to search the literature for instances in which epistaxis, generally a banal and simply bothersome affliction, had assumed fatal proportions. The search was well rewarded. It turned out that the two cases described above were illustrative examples of the pathologic events culminating in death from nosebleed: A review of the records of the U. S. Veterans Administration Hospital at Los Angeles from 1949 to 1958 disclosed two additional cases of fatal epistaxis.

Nosebleeds spare no age group. They occur in the toddler who falls and strikes his nose, in the nervous nose-picker ("epistaxis digitorum") and in elderly arteriosclerotic persons. The usual local causes are foreign body, instrumental or digital manipulation, blows to the nose and infections and tumors of the nasal chambers. The generalized conditions which can lead to nasal hemorrhage are blood disturbances, such as leukemia and polycythemia, and disorders of the coagulation mechanism, as well as rheumatic fever and scurvy. Increased venous pressure as from emphysema, prolonged coughing or straining and tumors of the neck and upper mediastinum have also been cited as causative conditions. The nosebleeds that physicians most often see in children and adults are those associated with blunt trauma to the nose or maladroit cleansing of the nasal passages. Nosebleeds among the elderly are distressingly frequent, usually arising from the posterior portion of the nasal fossa and generally defying etiologic diagnosis. In the older patients, epistaxis is often associated with increased arterial pressure and evidences of arteriosclerosis. The association may be fortuitous, however, for hypertension and medical sclerosis are recognized manifestations of the senium, and nosebleed is commonly seen in their absence.

Halberg[7] effectively summarized the etiology of epistaxis in his report of 212 cases at the Mayo Clinic. In that series, 50.9 per cent of patients were over 54 years of age and in approximately 80 per cent of them the bleeding ceased spontaneously.

In his diagnostic endeavors the clinician is limited to a search for the most common causative factors and must attempt to exclude potentially serious but undiscovered illness of which the nasal hemorrhage may be the first sign. When considering fatal epistaxis, we must pay particular attention to the nasal hemorrhage that follows a head injury. Here, the basilar skull fracture has a definite role in that the line of fracture may intersect the course of a major blood vessel with resulting hemorrhage into the nasal chambers, paranasal sinuses, eustachian tube or nasopharynx. Additionally, these fractures of the floor of the brain case are difficult to detect radiologically, often remaining undiagnosed until postmortem examination. As a general rule, the injuries that result in the fortunately rare instances of massive, life-threatening epistaxis are those in which the anterior ethmoid artery or the internal carotid artery is torn. Injury to the latter vessel is sometimes associated with disturbances of vision, of facial sensation or of pituitary function and is accompanied by premonitory aura or intracranial bruit.

REVIEW OF THE LITERATURE

In a review of the medical literature for the preceding 100 years, reports were found of a number of cases of fatal nosebleed. The first account of such a case was given by Levy[10] who, in 1896, described a 20-year-old white male miner who was struck on

the jaw in an occupational accident in Colorado. Immediately after the injury severe epistaxis developed. It recurred repeatedly and was associated with suppurative otitis media and mastoiditis. Thirty-eight days after the injury, loss of blood from a nosebleed was great enough to cause death. Autopsy showed a "septic fistula" between the middle meningeal artery and the eustachian tube in the region of the foramen spinosum.

Matthews[11] in 1898 briefly mentioned a case of spontaneous fatal nasal hemorrhage in a 19-year-old boy who had "bilious fever." In 1899 Dock[6] discussed a 37-year-old man who had a fatal nasal hemorrhage arising from an "endothelioma carcinomatosum" of the turbinated nasal bones.

Kummel[9] in 1928 recounted the case of a 23-year-old man who received a head injury in a motorcycle accident. The blow left him unconscious and bleeding from the nose, mouth and right ear. He recovered rapidly but thereafter had spontaneous, severe epistaxis at approximately two-week intervals, accompanied by severe temporal headache which was worse on the left side. Sixty-eight days after the injury, total amaurosis supervened in the right eye, which showed a pale disc and extremely narrow retinal vessels. Ten weeks after the accident, the patient suddenly had tremendous hemorrhage from the nose and mouth and died within several minutes. Autopsy indicated that the bleeding was a result of a fissure in the thin bony wall between the sphenoid sinus of the internal carotid artery which communicated with a small laceration of the artery itself. This situation had led to the formation of an aneurysm of this vessel which produced pressure upon the second branch of the trigeminal, the sphenopalatine and, presumably, the optic nerves, resulting in the temporal headaches and blindness.

Bonnet,[3] in 1935, presented a similar instance of traumatic aneurysm of the internal carotid artery within the sphenoid sinus in a 31-year-old woman who three months earlier had had a fracture of the floor of the anterior fossa from a blow on the frontal region. Epistaxis which occurred immediately following the injury was treated by ligation of the external carotid artery, without success. Within three months of the injury, the right eye had become blind and the oculomotor nerve on that side had become paralyzed. Angiograms showed an intrasphenoidal carotid aneurysm. Ligation of the right internal carotid artery did not prevent a fatal nasal hemorrhage.

Bean,[1] in 1937, in a study of conditions which predisposed to myocardial infarction, cited one case in which severe epistaxis had led to a profound anemia and death. At postmortem examination, a fresh myocardial infarction was found. He also reported a case of fatal myocardial infarction following hemorrhage from a peptic ulcer, as well as two cases of infarction in patients who had profound anemia.

In 1939, Davis[5] recorded the case of a 17-year-old boy who was struck in the nose by a fist and received fractures of the nasal bones and the nasal processes of the maxilla. This was attended by severe epistaxis which eluded all attempts at control short of ligation of the major vessels and caused death on the night of injury. Postmortem examination showed the anterior ethmoid artery to be ruptured.

Rasquin,[13] in 1949, described his experience with a 46-year-old farmer who, for two years, endured recurrent epistaxis preceded by an aura of painful pulsations and a "heavy feeling" in the left temporal region. The nosebleeds were as remarkable for their brevity as for their severity. At one point the patient lost 950 cc. of blood in just 20 seconds. Exploration of the left maxillary sinus, ligation of the internal maxillary artery in the pterygomaxillary fossa, ligation of the external carotid artery and the application of intranasal radium therapy did not halt the episodes of nasal hemorrhage. As the bleeding continued, hemiplegia developed and the patient died two days later. Postmortem examination was not performed, but the author suggested the possibility of an aneurysm of the internal carotid artery as the underlying cause of the hemorrhages.

Bourdon,[4] in 1952, reported the case of a 30-year-old man with spontaneous persistent epistaxis despite the appearance of excellent general health. Within a few weeks fever and leukocytosis developed; the peripheral blood assumed the appearance of leukemia. Seven days later the epistaxis ceased, but on the following day the patient died. Postmortem examination was not reported.

Sigiuer,[15] in 1954, told of a 50-year-old white man who had the florid complexion typical of alcoholics and hepatosplenomegaly suggestive of Laennec's cirrhosis. The patient steadfastly denied the use of alcohol in any form or quantity, stating that his father had died a drunkard and as a result he himself had formed a lasting antipathy to liquor. The patient had been plagued with nosebleeds since the age of six years. Further, his father, brothers and two nephews had the same affliction. One brother died of spontaneous hemothorax, secondary to a pulmonary arteriovenous aneurysm. Upon careful examination of the patient, multiple, mucosal telangiectases were observed in the oral and nasal cavities. Despite vigorous local treatment, the patient died of a spontaneous nasal hemorrhage, a consequence of Osler-Weber-Rendu syndrome.

Hrenoff,[8] in 1954, wrote of his experiences in the treatment of a 61-year-old hypertensive man who had severe recurrent epistaxis that reduced the hemoglobin content to 7 gm. per 100 cc. of blood. The

patient was given Bistrium® (hexamethonium) to reduce the arterial pressure, but the patient died that night. The Bistrium® combined with the anemia due to blood loss caused fatal circulatory collapse.

Seftel,[14] in 1959, discussed epistaxis resulting from rupture of an intracranial aneurysm and presented the case of a 20-year-old man who died of exsanguination from this cause five months after receiving a head injury. He had had repeated, small nosebleeds in the interim and the fatal hemorrhage was from the nose and mouth.

From these recorded experiences, we see that death from nasal hemorrhage may be the result of simple exsanguination or it can represent the combination of a preexisting limited circulatory reserve upon which profound anemia is suddenly imposed. Loss of blood great enough to cause death may follow a blow to the head or may be associated with defective blood coagulation, with nasal hemangiomata or with idiopathic aneurysm of the internal carotid artery.

DIAGNOSTIC PROCEDURES

A careful history is always desirable but not always obtainable when dealing with a person whose overwhelming concern is the arrest of nasal hemorrhage. Once the bleeding has been controlled, it is of importance to search the patient's history for possible clues as to the genesis. Questions should be asked that will bring to light any diseases of the cardiovascular system, the blood-forming tissues and the blood-coagulating mechanism. The presence of a neoplasm in the nose or paranasal sinuses must be suspected, and the patient should be questioned carefully with regard to recent head injury and nasal trauma. The interrogator is wise also to pay close attention to symptoms suggesting coronary artery insufficiency or cerebrovascular disease, for these conditions can assume critical importance in the presence of sudden severe blood loss.

Of utmost importance in the definitive treatment of severe epistaxis by cautery, packing or ligation is the accurate localization of the source of the bleeding. For this purpose, a neurosurgical suction tip supported by a vigorous suction machine, an optically accurate head-mirror and a strong source of illumination are desirable. The importance of accurate identification of the bleeding area cannot be overstressed, particularly in cases in which ligation of the major vessel appears necessary; for the decision concerning which vessel to ligate rests largely upon this observation. As a matter of routine, determinations should be made of the hemoglobin mass, of bleeding and clotting time and prothrombin time and the concentration of platelets. Morphologic studies of the blood should be carried out.

THERAPY

The most desirable methods of arresting nasal bleeding are those that compress or destroy the bleeding vessel yet inflict a minimum of injury to the surrounding structures and cause the least discomfort to the patient. Precise, firm packing over the offending vessels, either alone or in combination with electrical or chemical coagulation of the bleeding point, will control nosebleed in most cases. The details of various methods of local therapy have been splendidly presented by Ogura[12] and Beinfield.[2]

In general the most troublesome nasal hemorrhages arise from the relatively inaccessible regions of the olfactory cleft and the posterior choanae. In these cases cauterization is often impossible and bleeding cannot be controlled by anterior packing. In such circumstances a posterior choanal or nasopharyngeal pack may be required, not once, but repeatedly. Various forms of systemic therapy, including administration of vitamins C, K, and P, together with estrogenic substances and pituitrins, have been recommended.

While centering attention on the source of bleeding, the physician must be mindful of the effect of blood loss upon the patient, watchful for signs of approaching hypovolemic shock and ready to order intravenous infusion of plasma expanders or whole blood. It is not the presence of blood in the nose but the lack of blood in the vascular system that causes death.

Generous sedation optimally in the form of morphine sulfate is both the gesture of kindness and an effective hemostatic maneuver. Anxious, tense patients bleed more freely, probably as a result of the elevated arterial pressure in combination with the nasal vascular engorgement seen in emotional stress. Frequently overhearing a telephoned order for morphine sulfate will stop the bleeding before the physician arrives on the ward.

Appropriate surgical treatment of severe nosebleed includes a number of techniques. One is sometimes required to reduce a severely deflected nasal septum that is hiding the source of the bleeding. Should the bleeding point lie upon the septum itself, the scarring that follows the operation will often permanently obliterate the vessel. In cases of posterior epistaxis, it is sometimes expedient to infract the inferior turbinate on the side of the bleeding, thus obtaining clear access to the posterior portion to the inferior nasal meatus, the most common site of posterior nasal hemorrhage. Until recently ligation of major arteries for the control of epistaxis had been regarded as a last resort, to be used only when the hemorrhage assumed life-threatening proportions. The earliest ligations for this purpose were

performed upon the common carotid artery. The mortality from this procedure relegated it to the hands of a surgeon "at bay." Today, the external carotid artery and the anterior ethmoid artery are the vessels most commonly interrupted in the treatment of severe nasal bleeding. The techniques of ligation employed upon these vessels carry with them a very small morbidity and no mortality whatsoever, as far as has been reported. The operations can be performed under local anesthesia and leave a minimum of external scarring. When the point of origin of the nosebleed can be accurately localized to the areas subserved by either of these vessels, arterial ligation is followed by prompt and permanent cessation of epistaxis.

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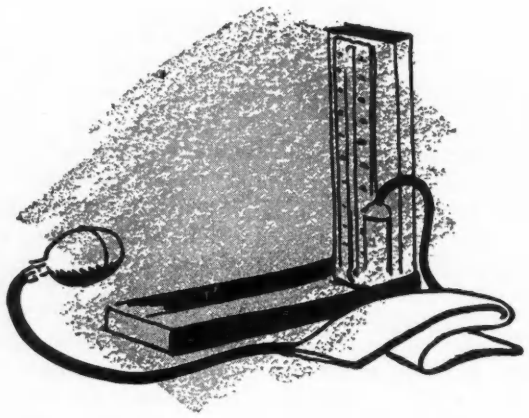

Clinical Experience With Fluether* Anesthesia

R. W. M. BETHUNE, *M.D.*, and HENRY UPHOLT, *M.D.*, Los Angeles

• **Fluether, an azeotropic mixture of two volumes of Fluothane® and one volume of diethyl ether, will provide satisfactory surgical anesthesia in a range of vapor concentration from 0.5 per cent v/v to 4.0 per cent v/v. The agent was used in all age groups and in all patient physical states in approximately a thousand cases.**

To date there have appeared no contraindications to the use of fluether anesthesia.

Bradycardia is common during induction but is easily reversible by use of belladonna compounds.

During maintenance of anesthesia, significant muscular relaxation is provided, without evidence of electrocortical depression.

Respiratory depression accompanies a maintenance level of anesthesia.

Recovery after anesthesia with fluether is rapid but tranquil. Considerable analgesia persists during recovery.

THE ELUCIDATION of the anesthetic properties of Fluothane® by Raventos[6] in 1956 marked the introduction of one of the most potent and versatile agents into modern anesthesia. There is no doubt that Fluothane® is already firmly established as a major anesthetic as evidenced by the thousands of reported cases in which it was used, and undoubtedly many more thousands unreported. In 1958 Hudon,[4] reporting on the use of a mixture of Fluothane® and diethyl ether, suggested that the clinical advantages of this mixture included ease of management, less depression of blood pressure and of respiration as compared with pure Fluothane® anesthesia. This mixture of two volumes of Fluothane® and one volume of diethyl ether is described as an azeotrope although it more closely resembles a compound in the liquid state, vaporizes in a fixed ratio identical to that of the liquid residue—that is, two volumes of Fluothane® vapor to one volume of ether vapor. For ease of reference, this mixture is termed "fluether."

Our use of fluether was undertaken not to assess a substitute or alternative for any existing agent but to evaluate this mixture clinically as an additional anesthetic agent. To assess fluether clinically it must be compared under conditions similar to those in which existing anesthetic agents are used, although purposeful comparison is not the basis of this report. It is the intent of our anesthesia method when using any agent to provide only a light level of anesthesia and to secure muscular relaxation as needed by supplemental use of nonanesthetic relaxant drugs. It is rarely, if ever, our aim to attempt to provide both analgesia and relaxation with one drug or agent. We believe that true anesthesia of sufficient depth to provide profound muscular relaxation will provoke more severe physiological changes of a deleterious nature than will the so-called "light anesthesia" plus relaxants.

Material

Fluether was used in approximately a thousand cases in a period of 14 months. Our clinical experience covers all age groups (the youngest patient, 36 hours) and all physical states and circumstances of operation, from "healthy, elective" to the "critical, emergency." With the exception of cardiopulmonary by-pass procedures and obstetrical procedures, surgical operations of all types are included in this report.

Apparatus

In most of the early cases a Fluotec® vaporizer was used to deliver a controlled vapor concentration to a non-return circuit. As experience was gained, other vaporizers and other circuits were employed. At present, fluether is administered in semi-closed, closed, and non-return circuits using a variety of vaporizers, such as the Ohio Vernitrol,® Foregger Copper Kettle,® Ohio Fluothane Vaporizer® and Fluotec.® It should be noted that all these circuits are assemblies of the "out-of-circle" type. Fluether has also been used by catheter insufflation for bronchoscopy and for laryngoscopy.

Using assemblies of the "out-of-circle" type as mentioned above, the expression of per cent fluether vapor as it appears in subsequent paragraphs refers only to the concentrations delivered from the anesthesia machine to the patient breathing circuit. When a closed or semi-closed breathing circuit is employed, this measured vapor concentration will be altered by the admixture of the patient's exhalations, and the actual inhaled gas will be of unknown vapor concentration.

*An azeotropic mixture of two volumes of Fluothane® and one volume of diethyl ether.

From the Division of Anesthesia, UCLA Medical Center (Bethune) and Morningside Hospital, Los Angeles (Upholt).

Presented before the Section on Anesthesiology at the 89th Annual Session of the California Medical Association, Los Angeles, February 21 to 24, 1960.

METHODS AND CLINICAL OBSERVATIONS

Induction

The induction is smooth and rapid, both in children and in adults, although in the latter patients a sleep dose of thiobarbiturate is usual. There is noticeable absence of salivation during induction, a circumstance well appreciated in pediatric anesthesia. There is no respiratory rejection of the vapor by the patient and maximum induction concentrations (up to 4 per cent v/v or 5 per cent v/v) can be delivered after a few breaths without irritation or breath holding. Intubation can be performed easily after a fluether induction, pharyngeal and laryngeal reflexes being remarkably quiescent. It is more usual to use succinylcholine to facilitate direct oral intubation in both adults and children, and there seems to be no reason to withhold this drug in any case in which its use might obviate even a remote possibility of injury.

Induction concentrations of 3 per cent volume-for-volume to 4 per cent v/v are delivered from the vaporizer with a half-and-half mixture of oxygen and nitrous oxide at a total diluent flow rate of 4 to 5 liters per minute. This induction concentration of vapor and diluent flow is continued for about ten minutes, provided the blood pressure change is not extreme. As with Fluothane® anesthesia, the calm, quiet induction of fluether may give a false impression of depth of anesthesia and too early stimulation will provoke a very vigorous response. During induction some pulse rate slowing is fairly common; atropine or other belladonna compounds are used early and in sufficient doses to provide a suitable increase in heart rate.

During some phase of the induction, frequently in the early minutes, there will usually be mild to moderate depression of the blood pressure as compared with the pressure before anesthesia. Abrupt and profound hypotension as seen occasionally with Fluothane® induction has not been apparent when using fluether in comparable concentration. The occurrence of moderate depression of blood pressure signals the end of delivery of higher fluether concentrations but does not, per se, indicate anesthesia of sufficient degree to permit surgical stimulation; rather, this agent, as indeed any agent, must be delivered over a variable but sufficient time before adequate anesthesia will result. This blood pressure depression is used only as a guide for the proper setting of the vaporizer control to maintain the desired depth of anesthesia. The limit of the blood pressure depression is planned to be about 30 mm. of mercury below the pre-anesthetic reading in normotensive subjects. If this limit is considered excessive for any patient—as it might be for, say, a patient in shock or one with heart disease or hypertension—or if the limit is grossly exceeded, reduction in the concentration of fluether vapor delivered will readily reverse the decrease in pressure. The intravenous injection of atropine at this time will also help to elevate the blood pressure. Rarely, have we found vasopressor agents necessary, and use of them is becoming even less frequent as we gain in experience in the use of this anesthetic. After the period of induction at high total gas flows, there is adequate denitrogenization, and reduced total flow for closed circuit technique can be administered if desired.

Maintenance

Maintenance concentration of fluether vapor delivered into circuit will vary from less than 1 per cent v/v to about 3 per cent v/v. The most reliable guide to this concentration is the level and stability of the blood pressure; as previously mentioned, an acceptable vapor concentration usually results in a stable but slightly depressed blood pressure. As in anesthesia using any agent, if maintenance doses of fluether are inadequate, stimulation will result in an elevation of the blood pressure.

When using the Fluotec® vaporizer the total gas flow, whether oxygen alone or a mixture of nitrous oxide and oxygen, is not reduced below one liter per minute, because of variability of the vapor concentrations with this instrument at lower flow rates. With the "Copper Kettle"® and vaporizers of similar type, lower total flow rates are sometimes used, although no persistent attempt is made to provide completely closed-circuit anesthesia in the majority of cases.

Repeated electroencephalographic recordings in cases in which this method of maintenance was used resulted in electrocortical patterns indicative of "light" anesthesia—that is, fast activity (15 to 20 cycles per second) with voltages up to 100 microvolts and only occasional areas of mixed fast and slower patterns. There has been no "burst suppression" attributable to the level of anesthesia. No respiratory stimulation in depth or minute volume has been consistently observed during fluether anesthesia. Light anesthesia with adequate pulmonary ventilation provide optimum conditions in respect to patient physiological response to anesthesia.[2,3] Fluether anesthesia is accompanied by some respiratory depression and it is our invariable habit to assist or control the ventilation during the course of the anesthesia.

Light anesthesia with fluether will provide appreciable muscular relaxation, usually adequate for major portions of pelvic laparotomy, genito-urinary procedures and other surgical adventures not demanding the cadaveric conditions requested for complicated upper abdominal procedures. If relaxation of a greater degree than that provided by the fluether anesthesia is required or requested, this

additional relaxation is accomplished by the use of succinylcholine, either by intermittent dose method or by continuous drip, depending on the surgical situation. The level of anesthesia is not changed to attempt to provide relaxation in these circumstances; our preference is to maintain a steady state of anesthesia and supplement with succinylcholine as required. The choice of succinylcholine as the relaxant is based on the circumstances involved in its use; namely, requirements for relaxation over and above that which is intrinsic in the anesthesia will be discontinuous as the surgical demands vary, and it seems that a controllable, short-acting agent would most specifically meet the needs of the situation. There has not been any case of "prolonged" apnea with the use of fluether and succinylcholine in this series, even though in some of the cases anesthesia was maintained for 8 or 9 hours.

Termination

When the anesthesia has been of such duration that a stable plasma level of drug can reasonably be assumed (that is, when there is equilibrium with a fixed vapor concentration in inspired air), it is desirable to discontinue the delivery of vapor about thirty minutes before the operation is expected to be finished. This early but calculated cessation of vapor administration will avoid the apparently prolonged periods of recovery that have been reported.[1] Although recovery time is a very difficult parameter to evaluate, and no attempt has been made to measure this factor accurately in this series, certainly in cases where the anesthesia has been brief and the operation uncomplicated the recovery time is as short as could be desired. In cases in which the anesthesia is prolonged or the operation of great magnitude, it has been impossible to correlate recovery time with anesthesia method or agent. The emergence from fluether anesthesia is smooth, quiet, and unattended by the abrupt, excited awakening sometimes seen after Fluothane® anesthesia, especially in children. We believe that this tranquil recovery after fluether is indicative of a significant degree of postoperative analgesia. In a few patients observed in the recovery room the incidence of nausea and vomiting was greater after fluether than after Fluothane,® but no statistical analysis on this point was attempted. The occurrence of nausea and vomiting is the common episode coinciding with first few minutes of waking and is rarely repetitive. Persistent vomiting did not occur in this series.

DISCUSSION

The azeotropic mixture of two volumes of Fluothane® and one volume of diethyl ether has been used satisfactorily for over a year in many varieties of circumstances.

In the decision as to what method and agent to use for anesthesia, certain standards should be observed for patient safety and satisfaction as well as for practical ease of management for the anesthesiologist. Among these standards are:

1. Agents should be applicable for use in a vast majority of cases, not restricted by age or physical condition of patient or type of proposed operation.

2. The same agent should be applicable for both induction and maintenance of anesthesia. This is especially referable to pediatric anesthesia.

3. The anesthetic agent and method should not be objectionable to the patient.

4. The anesthesia should be adequate without sacrifice of acceptable oxygen concentrations.

5. The anesthesia should be satisfactory but not at the expense of secondary physiological derangements.

6. Because of the prevalence of various electrical devices in the operating rooms, the preferred anesthesia should be nonexplosive.

At the present state of experience with fluether anesthesia, it is considered that this agent does, in fact, satisfy these standards for use.

There have been no circumstances so far encountered that prohibit or restrict the use of fluether anesthesia. The induction of anesthesia is smooth, rapid, and pleasant for the patient; emergence is quiet and not so abrupt as to create excitement. Significant analgesia appears to accompany the emergence, lessening the need for sedation in the immediate postoperative period. Maintenance of anesthesia can be provided with fluether vapor concentrations of from less than 1 per cent v/v to 3 per cent v/v and therefore the diluent can contain any portion of oxygen that is desired up to approximately 96 or 98 per cent. Fluether vapor is compatible with all other anesthetic agents in common use today, also with all relaxants although some restrictions pertain to the use of d-tubocurare because of the additive effects of Fluothane,® ether and the curare.[7] The vapor is stable in the presence of soda lime. Severe hypotension has not occurred in degree or frequency to compare with similar experience using Fluothane.® Possibly this reflects the stimulating effect of the ether fraction on the autonomic system and partial compensation of the pronounced hypotensive properties of Fluothane.®[5]

In the range of anesthesia concentrations, the vapor is nonexplosive and therefore there is no restriction on the use of any electrical devices.

The concomitant use of fluether anesthesia and local infiltration with solutions containing epinephrine is discouraged, although this has occurred inadvertently on several occasions without ill effect.

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ANNUAL SESSION PROGRAM

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MARCH ISSUE

CASE REPORTS

Occlusion of the Middle Cerebral Artery in Children

A Report of Two Cases

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SPONTANEOUS OCCLUSION of major intracranial arteries is relatively common in adults but has been considered to be rare in infants and children. Many of the reported instances of cerebral vascular occlusions in childhood have occurred in patients with cardiac disease.[1,9] The clinical entity of acute infantile hemiplegia has been assumed to be due to occlusions of cerebral vessels, although verification of this has been infrequent.[5] A recent report by Banker,[1] based on postmortem material, suggests that cerebral vascular disease in childhood is more common than previously suspected.

The increased use of cerebral angiography in children, as well as careful postmortem examination, has contributed to the understanding of these conditions. Fisher and Friedmann[4] reviewed the literature, found reports of 16 patients aged 15 years or less with thrombosis of the internal carotid artery, and reported one additional case. Stevens[8] reported upon three children with internal carotid artery occlusion, two of whom were included in the previous compilation.[4] Duffy and coworkers[3] de-

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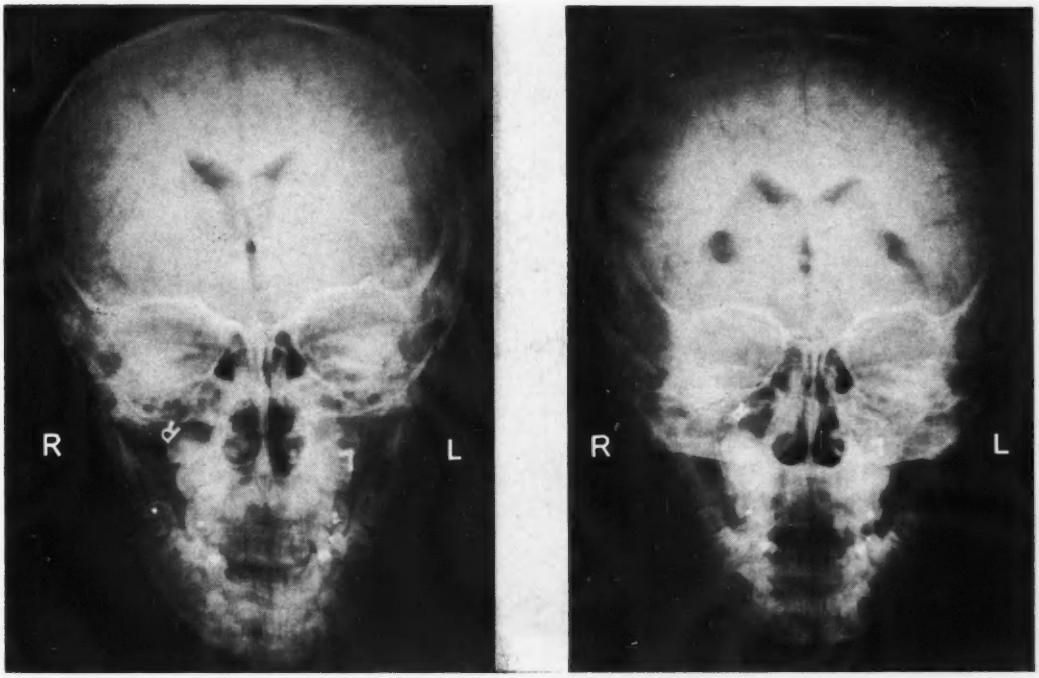

Figure 1 (Case 1).—*Left*, antero-posterior encephalogram and, *right*, postero-anterior encephalogram showing right lateral ventricle slightly larger than left.

scribed the pathological findings in a 16-month-old child with spontaneous thrombosis of the intracranial portion of the left internal carotid artery.

Following are reports of two children with complete occlusion of the middle cerebral artery which was demonstrated by carotid angiography.

REPORTS OF CASES

CASE 1. On May 14, 1951, a right-handed white boy almost six years of age had an episode of incoherent speech, fecal incontinence and left hemiparesis, which disappeared in 15 to 20 minutes. Two days later a transitory period of confusion and

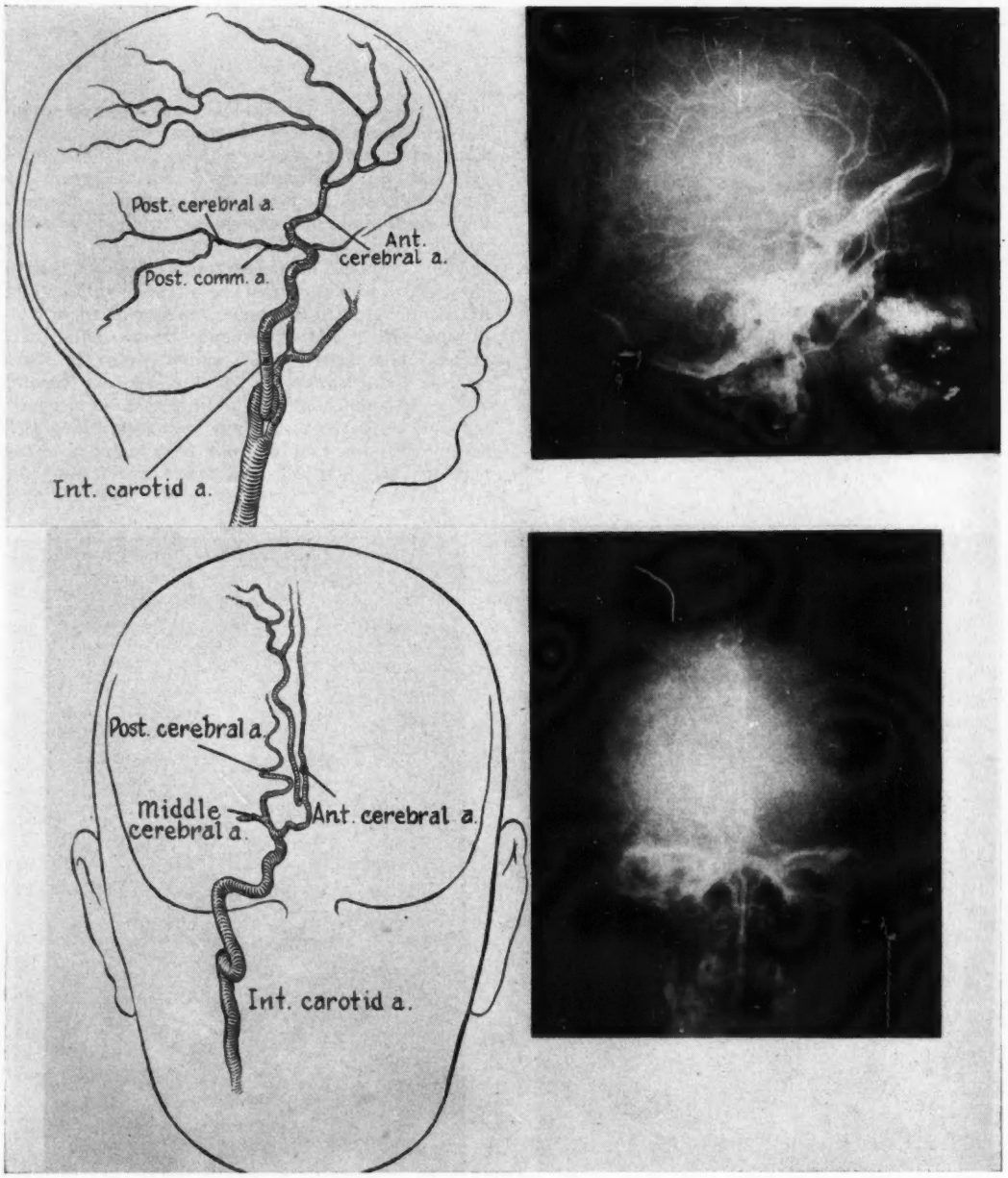

Figure 2 (Case 1).—Lateral carotid and antero-posterior carotid arteriograms with artist's drawings to emphasize the cerebral arteries. The right middle cerebral artery is occluded near its origin.

lassitude occurred. When the child was seen in the clinic on May 18, 1951, complete neurologic examination was within normal limits. An electroencephalogram was reported to show generalized dysrhythmia of nonspecific type with a suggestion of localized abnormality in the left temporal region. No abnormalities were seen in roentgenograms of the skull. While awaiting admittance to hospital, anticonvulsant medication was started on the assumption that the episode had been a convulsive seizure. However, 11 episodes of left-sided paralysis occurred before the patient was put in hospital some two weeks after the first attack. The severity of the attacks varied; during the most severe one, complete leftsided paralysis lasted for eight hours. In addition, a progressive personality change occurred and the child became irritable, aggressive and restless.

Birth and development had been normal. At the age of three years the child had an illness characterized by sore throat, polyarthritis and intermittent fever for several weeks. Complete details of this illness were not available but it was thought to have been acute rheumatic fever. A physician had noted a heart murmur for several months after this illness. The child's kindergarten teacher thought that his motor coordination was slightly below normal.

When admitted to the hospital on June 1, 1951, the child seemed alert and cooperative. The cardiac rate was regular and there was a Grade II systolic murmur which was loudest at the apex. Slight left lower facial under-activity and mild hypoactivity of the reflexes in the left lower extremity were observed. An electroencephalogram was reported to show high potentials and irregularity in the temporal and postcentral regions on the right, high potentials in the right low frontal and occipital areas and relatively low potentials in the right precentral region. There was spiking in the right temporal and post-temporal areas.

There were positive reactions to Kolmer and Kahn tests for syphilis on June 4; on July 10 the result of a Kolmer test was negative.

On June 2, 1951, with the patient under general anesthesia a pneumoencephalogram was made. The spinal fluid was under normal pressure and contained 45 cells per cubic millimeter, of which 12 per cent were polymorphonuclear leukocytes and 88 per cent lymphocytes. The spinal fluid contained 19 mg. of protein, 705 mg. of chloride and 22 mg. of sugar per 100 cc. Spinal fluid serologic study was negative for syphilis and the colloidal gold curve was within normal limits. The right lateral ventricle was found to be slightly larger than left (Figure 1). Following the pneumoencephalogram there was persistent left hemiparesis with an extensor plantar response on the left.

On June 8, bilateral carotid angiography demonstrated complete occlusion of the right middle cerebral artery (Figure 2).

On June 13 a pediatric consultant noted a grade II systolic murmur in the pulmonic area and a grade I systolic murmur at the apex, without diastolic components. The heart was not enlarged and regular sinus rhythm was present. The history suggested rheumatic fever with some mitral valve involvement, although there was no indication of active rheumatic fever at this time. Psychometric examination did not indicate intellectual deterioration or impairment.

The patient was discharged June 15, 1951, with instructions to take 100 mg. of nicotinic acid three times daily.

He was seen periodically thereafter, and slow improvement in the left hemiparesis was noted. A psychometric examination in November, 1951, was reported as showing an intelligence quotient of 80, and some deficiency was noted in number concept and inability to make abstract comparisons on the Wechsler Children's scale.

In January, 1953, the patient still had moderate spastic left hemiparesis, with the left upper limb more involved than the lower, and a hemiparetic gait. The left plantar response was extensor.

A report in 1957 stated that his gait was "acceptable," but he still required a night splint and stretching exercises for a shortened heel cord. The left upper extremity was used very little.

CASE 2. The patient was a four-year-old girl who was admitted to San Francisco General Hospital on March 14, 1959. Birth and development had been normal and she had been speaking in full sentences. About four weeks before admission, following a respiratory infection, she seemed irritable and had several temper tantrums, and two days before admission her gait appeared to be clumsy. During the night before admission, the patient fell from her bed several times but did not lose consciousness. In the morning she was unable to stand or speak, and when she attempted to swallow food it ran out of the right side of her mouth. Because of the history of falling from bed, the possibility of an intracranial hematoma was considered.

At hospital admission the child appeared to be aphasic and drowsy. There was right hemiparesis, right hemihypalgesia and apparent right homonymous visual field defect. Spinal fluid pressure and cell count were within normal limits. Carotid angiography demonstrated complete occlusion of the left middle cerebral artery (Figure 3).

Results of routine blood cell count, urinalysis and serological tests for syphilis were within normal limits.

There was gradual improvement in speech and in the strength of the right extremities. When examined four and a half months after the onset of hemiplegia, the patient walked well with a slightly hemiparetic gait. There was tightness of the right heel cord. She was speaking clearly.

DISCUSSION

The descriptions of intracranial arterial occlusions in childhood have been based, to a large extent, on pathological examination in fatal cases. Even in these studies, the site of occlusion is not

always identified, although the area of infarction appears to correspond to the distribution of a specific cerebral vessel.[9]

The sudden onset of hemiplegia in a child with congenital heart disease requires little comment. Other causes of cerebral arterial occlusions in childhood include many of the severe infectious diseases,[2,5] infections involving the vessel wall,[1,2] dissecting aneurysm of a cerebral artery,[7] congenital syphilis,[2] and atherosclerosis.[5]

In the two cases reported here complete occlusion of the middle cerebral artery was demonstrated by

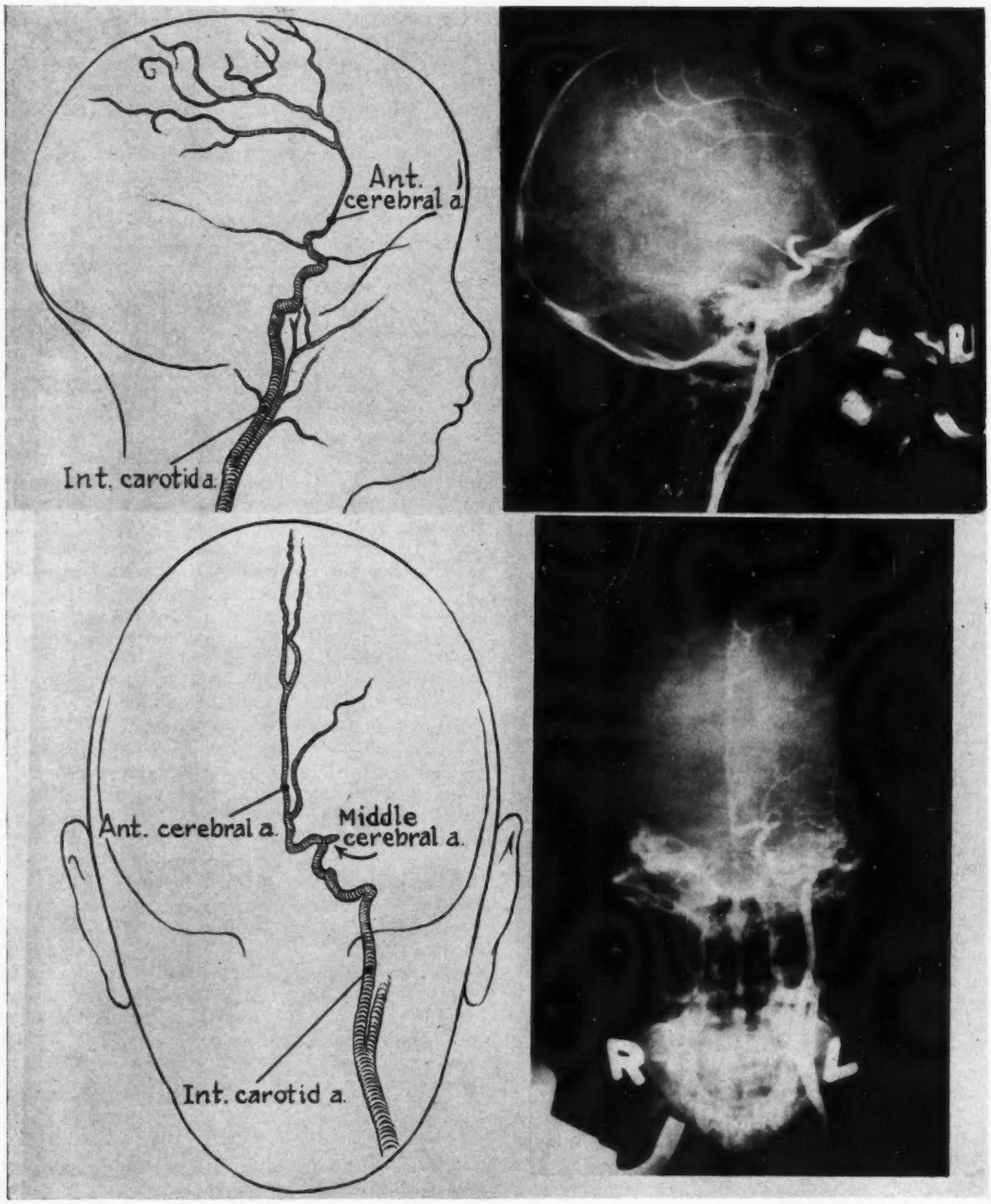

Figure 3 (Case 2).—Lateral and antero-posterior carotid arteriograms and artist's drawing. The left middle cerebral artery is occluded near its origin.

carotid angiography. In the absence of evidence of a primary source for an embolus, it is assumed that the occlusions were thrombotic in nature. In Case 1, the occlusion may have been related to rheumatic fever. In Case 2, there was no clinical evidence of any systemic disease.

The manner of onset of the symptoms in these two cases is noteworthy. In both instances premonitory signs were present for some time before the onset of permanent hemiparesis. In Case 1, the transient episodes of hemiparesis were at first interpreted as focal convulsive seizures with postconvulsive hemiparesis. This manner of onset is not unlike that seen in certain adult patients with occlusion of the internal carotid artery.[6] In Case 2, the premonitory signs were personality change and unsteadiness of gait.

From the evidence available in these cases, one cannot say whether the intermittent premonitory signs were due to gradual narrowing of the vessel before occlusion or to variations in collateral circulation after occlusion had been established. In Case 1, the relationship of onset of permanent hemiparesis to the pneumoencephalogram should be noted.

SUMMARY

In two children, aged six and four years, hemiplegia developed after several weeks of premonitory symptoms. The diagnosis of occlusion of the middle cerebral artery was established by carotid angiography. Neurological recovery was not complete in either case.

ADDENDUM: Taveras and Poser recently reported angiographic demonstration of occlusion of the middle cerebral artery in three children with infantile hemiplegia (Taveras, J. M., and Poser, C. M.: Roentgenological Aspects of Cerebral Angiography in Children, Amer. J. Roentgen., Rad. Therapy and Nuclear Med., 82:371-391, Sept. 1959).

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Hydatid Cyst in Muscle of the Thigh

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HYDATID DISEASE, also called echinococcosis or echinococciasis, is caused by the larval form of the tapeworm, *Taenia echinococcus granulosus*. The highest incidence is in sheep and cattle raising countries, particularly in Australia, Central Europe, New Zealand, Asia, Iceland and the countries bordering the Mediterranean. A few cases are reported each year from the southwestern areas of the United States.[5]

The disease is disseminated by the feces of infected canines. Dogs, foxes and wolves act as the definitive host. The intermediate hosts are usually sheep, hogs and cattle. The adult worm of *E. granulosus* is 3 to 5 mm. in length, and consists of a scolex, or head, with four suckers and a rostellum with two rows of hooklets. The scolex includes the head and narrow neck and is subtended by three proglottids.[1]

Human infestation results from ingesting contaminated food or water, or from finger pollution whereby eggs of the worms are introduced into the digestive tract. These eggs hatch in the small intestine, and the embryo burrows through the mucosa into the mesenteric venous system, whence it is carried to the liver by the portal circulation. The liver traps most of the larvae; those that escape go to the lungs, where they may settle. The larvae that are not filtered out by the lungs will pass to the left side of the heart and to the general circulation. Once in the general circulation, they may seed in any organ or tissue of the body.

When a final destination is reached by the embryo, it develops into a cystic form. The cyst grows at a slow rate; in some cases the cyst may be as old as the patient, infestation having taken place in infancy. Some cysts attain tremendous size; seven to ten liters of fluid have been reported in the larger ones.[4,6] In addition to fluid in the cyst cavity, there are found brood capsules, scolices and daughter cysts. The hydatid fluid appears to be clear, but if it is permitted to stand, a sediment containing scolices will settle to the bottom. This sediment is the so-called "hydatid sand."

Rupture of the cyst either by trauma or inadvertently by surgical puncture will allow the hydatid sand to escape into the tissues and there produce secondary cysts. The other hazard of leakage from a cyst is that of anaphylactic reaction in the host, the cyst fluid being antigenic.

About 70 per cent of human hydatid disease occurs in the liver. Ten per cent of lesions are found in the lungs.[4] Hydatid cysts rarely appear in peripheral sites—muscles and bones.

This study was aided by a grant from the Isaac Brotman Foundation of the Culver City Hospital.

From the Departments of Surgery and Medicine, Culver City Hospital, Culver City.

Submitted July 26, 1960.

The Casoni intradermal test as modified by Dew, Kellaway and Williams[2] and the complement fixation test of Ghedini[3] and Weinberg[7] may be of positive diagnostic help. However, deterrents to the tests are negligible amounts of antigen that may be absorbed from the cysts, and the scarcity of fresh antigen in this country.

The only successful treatment is complete excision. If excision is incomplete, the remaining cyst may continue to develop. Chemotherapy is entirely ineffective.

The unusual location of the cyst in the present case, the large dimensions and the characteristic tumefaction in a person from an endemic region prompted this report.

REPORT OF A CASE

A 61-year-old white man, a grocer, was admitted to the Culver City Hospital on February 22, 1957, with a large, hard, painless lump in the right upper thigh. The lump had been present for four to six months and was becoming larger. It was not tender and there was no pain in the area. The patient had no history of any severe illness. Born in Greece, he had served in the Greek army during World War I, then had come to the United States. On later questioning, he denied any contact with animals such as sheep or dogs.

Except for a large, firm mass in the right thigh, no abnormalities were noted on physical examination. The blood pressure was 110/70 mm. of mercury and the pulse was regular at 80 per minute. Respirations were 20 per minute. The temperature was 98.6° F. There was no generalized enlargement of lymph nodes. The abdomen was soft and no masses or organs were felt. The mass in the thigh was in the upper medial third. It seemed to extend deep to the fascia lata and was movable but not tender. There was no discoloration of the skin over it and no bruit was heard.

Results of a serologic test for syphilis were negative. Erythrocytes numbered 4.6 million per cu. mm. of blood and hemoglobin content was 14 gm. per 100 cc. Leukocytes numbered 8,750 per cu. mm.—75 per cent segmented forms, lymphocytes 22 per cent, monocytes 1 per cent and eosinophils 2 per cent. Results of urinalysis were within normal limits. Cephalin flocculation was negative. Alkaline phosphatase value was 2.2 units. In roentgenograms of the pelvis, lumbosacral spine and chest, no evidence was seen of metastasis to the examined areas.

The preoperative impression was of muscle sarcoma or liposarcoma. A longitudinal incision was made in the upper medial aspect of the right thigh, and on separation of the muscles in this area, an irregular mass of yellowish, semi-liquid, semi-firm tumor was seen, extending from the upper medial aspect of the thigh inferiorly and posteriorly behind the femur. The tumor was approximately 15 to 20 cm. in width and depth and varied from 6 to 8 cm.

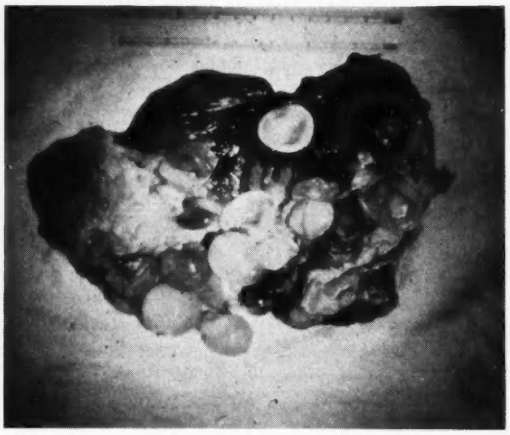

Figure 1.—Gross specimen, echinococcal cyst excised from thigh.

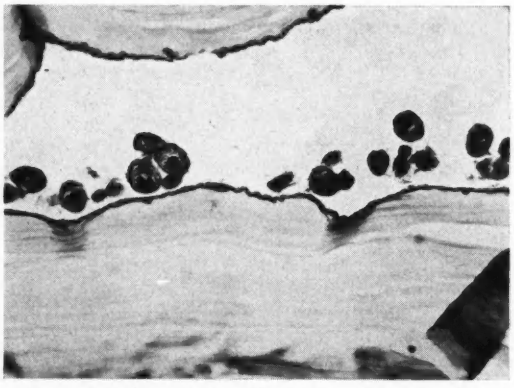

Figure 2.—Photomicrograph showing the cyst wall and echinococcus scolices (×160).

in thickness (Figure 1). The tumor mass was wound about the medial aspect of the thigh in such a manner as to extend from the antero-medial aspect inferiorly and posteriorly behind the femur and between the deep adductor muscles and the vastus medialis. It was removed by blunt and sharp dissection in the entire involved area. The wound was closed without placement of drains.

Pathologist's Report

The pathologist reported: The specimen was an ovoid mass, 16x12x7 cm., consisting of a fibrous sheath enclosing multiple cystic structures in necrotic fat. When the mass was incised, large numbers of soft, thin-walled, white cysts extruded. They lay in a stroma of fatty tissue, with yellowish chalky discoloration, suggesting fat necrosis. The cysts ranged in size from 1 to 3 cm. They contained clear but mucoid fluid and showed very tiny vegetations on the inner surfaces. Microscopically the cyst walls were seen as hyaline tissue, almost devoid of cells and forming anastomosing strands typical of the

lesion. Some of the sections showed attached scolices of Echinococcus. The peripheral capsular tissue in which some muscle fibers were attached showed chronic inflammation of mild degree, with a lymphocytic infiltrate. The diagnosis was multiple echinococcal cysts in muscle. (See Figure 2.)

The patient was discharged from the hospital five days after operation. He was examined occasionally in the ensuing three years and no evidence of recurrences was noted.

SUMMARY

Hydatid cyst (Echinococcosis) occurring in muscle is rare. Human hydatid infestation results from ingesting the hydatid eggs which subsequently hatch in the intestine and are finally carried to the liver by the portal circulation. Although the liver traps most of the larvae, those that escape settle in the lung, but some which are not filtered out in the lung will pass into the general circulation and seed in other tissue of the body.

The only successful treatment is complete excision, which was done in the case, reported herein, of an extraordinarily large cyst in an unusual location.

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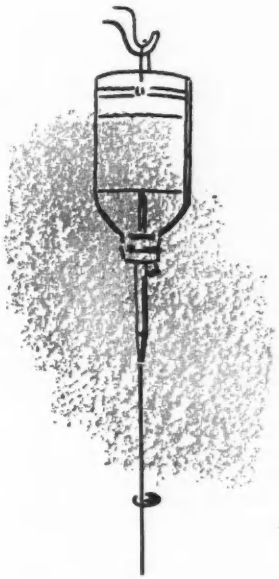

California MEDICINE

For information on preparation of manuscript, see advertising page 2

EDITORIAL

Legislative Time Again

IMMEDIATELY after the January 1 holiday the Legislature of the State of California went into session.

The session in this, an odd-numbered year, is wide open for the introduction of any kind of proposal the members of the Legislature may wish to advance. In the even-numbered years the state lawmakers consider only a budget and such specific matters as may be taken up in a special session called by the Governor.

If the pattern of past years is repeated now, we may expect to see some 6,000 bills introduced before the Assembly and the Senate. Of these, around 10 per cent, or about 600 measures, will have some direct or indirect bearing on the practice of medicine or the public health.

And if the precedent of earlier years is repeated, these bills will cover a wide variety of topics, will encompass both scientific and economic matters and will cause a large number of eyebrows to be raised in the medical and allied professions.

At this moment, one can only guess at the content of the many legislative proposals which are certain to be put into the hopper. Some members of the Legislature and some special interest groups, such as labor, have already made public announcements on the bills they intend to have introduced. Others have been more reticent, publicly, and will bring in their proposals without fanfare.

Specifically, labor has announced its intention of reviving the 1945 proposal of then Governor Earl Warren for the establishment of a state plan of medical care. Socialized medicine, it was called by physicians; not so, said the Governor, labor and other supporters of the plan.

The difference in definitions of "socialized medicine" was resolved in committee in the Assembly, when the Governor's plan failed to gain committee approval and just barely fell short of being brought out of committee onto the floor of the Assembly for debate.

For the record, it should be noted that the Governor tried again in 1947, and again in 1949, but his repeat efforts failed completely and without any of the furor which attended the 1945 trial.

Now, 16 years after the first try, labor says it will try again. If its efforts are in any measure successful, medicine will again be in for a fight, basing its resistance as it did before on the proposition that the socialization of medicine is simply a step in the socialization of all services and goods.

These observations are mainly academic at the moment, for the reason that labor's bill to socialize medicine has been introduced so recently that copies are not yet available for review and analysis.

A number of other bills have been announced publicly by their authors but have not yet been officially introduced. Here again, reliance cannot be placed on the public announcement; the bill itself, when and if introduced, is the item with which we have to deal.

As a result of publicity which legislators and others have received from public statements about what they say they *intend* to do, physicians in many parts of the state already are expressing concern over the potential harm to medical practice if some of the publicized proposals (not yet introduced as bills) should at last be submitted to the Legislature and adopted.

Policy in the California Medical Association has consistently been to refrain from any hurried conclusions or statements on legislation until (1) the bill has been introduced, (2) an opportunity is had to analyze it legally and for its effect, and (3) a position is taken on the measure by the Council.

This attitude is dictated by several sound considerations. In the first place, a public announcement of proposed legislation may represent nothing more than a trial balloon designed to find out who might be supporters and who opponents. Secondly, a public statement about proposed legislation may not reflect accurately on the measure as it is actually introduced. Thirdly, a quick stand announced on any

legislation may provide the author with a chance to compromise with announced opponents on minor matters in such a way that he can later claim to have consulted with critics of his proposal and to have revised his measure to meet their objections.

The California Medical Association has followed the legislative processes carefully for a number of years and has found that effective legislative activities require an active committee of members of the Association who can keep in touch with happenings in the Capitol, backed up by legal counsel versed in legislative draftsmanship and by representatives who can carry out the policy decisions reached by the Council on any given measure.

With this sort of representation, the Association is able to secure copies of measures placed before the Legislature, have them analyzed, secure a policy position from the Council and proceed to implement that policy.

Thus, in the present session of the Legislature, the watchword at this time must be "wait and see." Wait for the bill to be introduced; see what is in it and what effect it may have on the public health or on the practice of medicine.

Under this policy the Association will bide its time until the established procedures may be undergone. In legislation, as in so many other fields, timing is extremely important and hasty actions or decisions may well drain strength from the organization in advance of the real need for strength.

Obviously, the present legislative session will produce a number of proposals that will be considered inimical to the best medical interests of the public. Obviously, the California Medical Association will oppose all such measures.

It is fortunate that under the present rules of the Legislature, no action can be taken on any bill until 30 days has elapsed after its introduction, except for rare emergencies. This waiting period allows those interested in legislation to review and analyze each measure and determine what position to take on it. The Association will, of course, support those bills which appear good and oppose those seeming bad. The decision as to which category a measure fits can be made during the 30-day waiting period before even committee consideration can be given to any measure.

With health matters representing so large a segment of the total legislative effort in California, the proper and adequate representation of the medical profession and its allies is valuable beyond description. Such representation the C.M.A. now has and has had for some years.

Physicians who may become disturbed over headlines at this stage of the legislative meeting would be well advised to consider the procedures which the Association has found so useful over the years and which have proved their effectiveness.

In short, let's wait and see.

Letters to the Editor...

A FEW MONTHS AGO at a public riding stable in Los Angeles a man who probably did odd jobs for the stable owners gave a good swat to the rump of a sluggish horse. The horse was carrying an experienced hospital secretary who was an inexperienced horsewoman. The horse got the idea and set off on a fast trot. The rider soon fell off, landing solidly on her bottom. She wound up in the hospital in traction with diagnoses of back sprain and multiple bruises. When various staff doctors stopped in to see her, they asked how the accident happened. The next remark of each doctor, the secretary avows, was: "You're going to sue, aren't you?" Suit before sympathy. Now probably the girl has grounds for legal action, but my point is that the doctors should not have had pecuniary compensation as their first thought.

Too many doctors similarly want to magnify the injuries in automobile accidents to help the patient, his lawyer, and his doctors to get more money. The doctor, however, should think more of getting the patient well and quickly well, adding the appropriate admixture of encouragement and reassurance to his medical regimen.

There is a special reason for this cost conscious handling of even insurance cases that deserves repetition: This is the fact that it is the good and the innocent people who pay for the insurance awards. By the good and the innocent I am thinking especially of that segment of the public whom we doctors cherish as patients: our stable, middle income families who are not accident prone, who are careful, productive, conscientious citizens. They pay our bills cheerfully, and they have to pay all insurance costs directly or indirectly. The insurance companies by and large insure for themselves profits and growth by appropriate premiums. But the innocent public pays. Let us doctors do what we can to protect it and help it.

ARTHUR F. GREENWALD, M.D.

Los Angeles.

California MEDICAL ASSOCIATION

NOTICES & REPORTS

Council Meeting Minutes

Minutes of the 464th Meeting of the Council, San Francisco, Hilton Inn, December 10-11, 1960.

The meeting was called to order by Chairman Sherman in the Hilton Inn, San Francisco International Airport, on Saturday, December 10, 1960, at 9:30 a.m.

Roll Call:

Present were President Foster, President-Elect Bostick, Speaker Doyle, Vice-Speaker Heron, Secretary Hosmer, Editor Wilbur and Councilors MacLaggan, Wheeler, Todd, Quinn, O'Neill, Kirchner, O'Connor, Shaw, Rogers, Dalton, Murray, Davis, Miller, Sherman, Campbell, Morrison, Anderson and Teall.

A quorum present and acting.

Present by invitation were Messrs. Hunton, Thomas, Clancy, Collins, Marvin, Whelan, Edwards, Tobitt and Drs. Batchelder and Miller of C.M.A. staff; county executives Scheuber of Alameda-Contra Costa, Geisert of Kern, Field of Los Angeles, Grove of Monterey, Somerville of Napa, Bannister of Orange, Brayer of Riverside, Dochterman of Sacramento, Nute of San Diego, Neick of San Francisco, Thompson, Pearce and Monnich of San Joaquin, Wood of San Mateo, Lingerfelt of Fresno, Donovan and Colvin of Santa Clara, Funk of Solano, Bailey of Tulare, Brown of Sonoma; Dr. Malcolm Merrill, State Director of Public Health; Dr. Theo K. Miller, representing the State Director of Mental Hygiene; Mrs. Eunice Evans, State Department of Social Welfare; Messrs. Hassard and Huber of legal counsel; Messrs. Read, Salisbury and Fraser of the Public Health League; Dr. Robert Holmes, president of San Mateo County Medical Society; Dr. William Hickey, president-elect of Sacramento Medical Society; Messrs. Lyon, Niron and Paolini of California Physicians' Service; and Doctors Dan O. Kilroy, Donald Harrington, Francis E. West, Wayne Pollock and Bernard M. Kramer.

1. *Minutes for Approval:*

On motion duly made and seconded, minutes of the 463rd meeting of the Council, held October 22, 1960, were approved.

2. *Membership:*

(a) A report of membership as of December 8, 1960, was received and ordered filed.

(b) On motion duly made and seconded, 30 delinquent members whose dues have now been paid, were voted reinstatement.

(c) On motion duly made and seconded in each instance, 14 applicants were voted Associate Membership. These were: Charles J. Brooks, Harvey Robert Lerner, Jack Lieberman, Fred H. Mowrey, Stanley John Simon, Ethel Francis Young, Llewellyn Martin Jones, Los Angeles County; Daniel Allen Treat, Sacramento County; George J. Wiesseman, San Bernardino County; Lourdes O. Agcaoili, Seymour Fisher, San Francisco County; Isaac Persyko, San Joaquin County; Frederick P. Schnell, San Luis Obispo County; Donald M. Bramwell, Sonoma County.

(d) On motion duly made and seconded in each instance, five members were voted Retired Membership. These were: Jose Renato Lacayo, C. Morley Sellery, Reuel M. Spencer, Los Angeles County;

George M. Landrock, Marin County; B. J. Rohlfes, San Francisco County.

(e) On motion duly made and seconded in each instance, five members were voted a reduction of dues because of illness or postgraduate study.

3. *State Department of Public Health:*

Dr. Malcolm Merrill, State Director of Public Health, reported that additional funds were contemplated for the department's use in the field of alcoholic rehabilitation. He also reported that progress was being made in automotive exhaust control and that several departments of the state were conferring on a possible coordinated program of traffic safety.

4. *State Department of Mental Hygiene:*

Dr. Theo K. Miller reported that two units for concentrated mental health therapy are soon to be opened, one at University of California at Los Angeles and one at the Napa State Hospital.

5. *State Department of Social Welfare:*

Mrs. Eunice Evans, Assistant Director of the State Department of Social Welfare, reported that a survey of retail drug prices showed that the department is purchasing prescription drugs for its recipients at a cost somewhat less than is paid by the general public.

Mrs. Evans also reported that eight private and public rehabilitation centers had been approved, that additional centers would likely qualify for approval in the near future and that the rehabilitation program for the aged would start on January 1. She also reported that the health evaluation studies of the aged would start on February 1.

6. *Unfinished Business:*

(a) Dr. Sherman reported for the ad hoc committee to study the report of the Governor's Committee for Medical Aid and Health. This report to date is in outline form, in advance of the complete report. On motion duly made and seconded, it was voted to accept the ad hoc committee's report as a progress report.

(b) Dr. Todd reported that the committee to plan the 1961 Conference of County Society Officers had voted to recommend that the conference be scheduled for February 10 and 11 and that the president, president-elect and secretary and two members of the House of Delegates be invited from each society, their expenses to be met by the Association. On motion duly made and seconded, this recommendation was approved.

Dr. Harrington reported that the Commission on Medical Services also wished to invite two representatives of each society to attend this conference at the Commission's expense. On motion duly made and seconded, this proposal was approved. It was also agreed that representatives of other organizations might be invited as individuals but not as groups.

7. *California Physicians' Service:*

Dr. John G. Morrison, chairman of the board of California Physicians' Service, reported that there are now 14,462 physician members and that the organization's financial status is satisfactory. He also reported that progress is being made on studies covering payments for consultative services and podiatrists' services.

Dr. Morrison also discussed the MD-65 program of C.P.S. and suggested that the program be expanded, possibly to include major hospitalization coverage with a $500 deductible clause and a $5,000 maximum benefit. On motion duly made and seconded, it was voted to reactivate the Council's committee on MD-65, possibly with new members. This committee is to be named by the Chairman of the Council and the Chairman of the Committee on Committees and is to present a report at the next Council meeting.

8. *Report of the President:*

Dr. Foster reported on actions taken by the Committee for Emergency Action at a meeting held November 23, 1960. Principal item at the meeting was passage of a resolution urging the American Medical Association to assume leadership in investigating the feasibility of providing broad-scale health insurance coverage for all people under a voluntary program. On motion duly made and seconded, this report was approved.

9. *Report of the President-Elect:*

Dr. Bostick reported on the reactions he had received on some of his visits to county societies and urged that members of the Council work with their constituents for achieving an understanding that a public relations program was a most involved problem and that professional understanding of the goals of the profession was essential in this regard.

10. *Finance Committee:*

Chairman Heron reported for the Finance Committee on several items, as follows:

(a) The committee recommended against preparation of a mailing list using home addresses of members, because of the difficulties and cost involved. On motion duly made and seconded, this recommendation was approved.

(b) The committee recommended that the expenses of representatives to the Governor's Con-

ference on the Aging be paid where such representatives had been nominated by the Association but that the expenses of others not be met. On motion duly made and seconded, this recommendation was approved.

(c) On motion duly made and seconded, the committee's recommendation that four official representatives of the Association to the White House Conference on the Aging be reimbursed for their expenses was approved.

(d) On the committee's recommendation and on motion duly made and seconded, it was voted to approve the formation and operation of a publishing subsidiary for the distribution of pamphlets and other printed materials. The corporation, to be known as Six Ninety Three Sutter Publications, Inc., will be wholly owned by Trustees of the California Medical Association. On motion duly made and seconded, interfund transactions between this corporation and the Association were approved.

(e) On motion duly made and seconded, it was voted to reaffirm an earlier Council decision calling for the payment of a per diem to a specified committee chairman only for those days when the chairman was absent from his office because of an approved request from a component county society.

(f) On motion duly made and seconded and by a three-fourths recorded vote, it was voted to appropriate an additional $4,000 to cover the expenses of the 1961 Conference of County Society officers, to be held February 10-11, 1961.

(g) On motion duly made and seconded, it was voted to approve in principle the committee's recommendation that a program of retirement annuities and life insurance be instituted for the employees of the Association, utilizing group coverages for both parts of the program. The Finance Committee was directed to work out necessary details.

(h) It was reported that a study of the Association's finances revealed that to avoid the current borrowing of money each fall to meet current obligations the sum of $25 a member, as a one-time assessment, would be necessary.

11. *Committee on Committees:*

Dr. Bostick recommended for the Committee on Committees that Dr. John F. Murray be added to the Liaison Committee to the California Pharmaceutical Association and that Dr. Donald Pettit be added to the Committee on Scientific Work to succeed Dr. Thomas H. Brem, resigned. On motion duly made and seconded these appointments were approved.

12. *Project "Hope":*

Dr. Bernard M. Kramer of San Francisco, just returned from a month of service on the ship *Hope*, reported on his experiences and on the program being followed by the project in Eastern countries which have requested the project's services. On motion duly made and seconded, it was voted that the Council of the California Medical Association views with favor the activity of the ship *Hope* as a highly meritorious program of people-to-people medical care between the United States and other countries.

13. *Liaison Committee to the State Bar of California:*

A report from the Liaison Committee to the State Bar of California recommended that limited privileges should be accorded members of medical disciplinary committees in the exercise of their duties and that Mr. Hassard be instructed to draft suitable legislation for the approval of the State Bar and for introduction into the Legislature. On motion duly made and seconded, this recommendation was approved.

14. *Commission on Medical Services:*

(a) Several recommendations for the operations and procedures of mediation committees in the county societies were discussed and, on motion duly made and seconded, referred back to the Commission for further clarification and report.

(b) Recommendations of the Commission relative to the August 19, 1960, report of the Commission—C.P.S. Liaison Committee were disapproved on motion duly made and seconded, and the Liaison Committee report of August 19 was approved.

(The Commission on Medical Services report recommended referral of House of Delegates resolutions No. 57, No. 75 and No. 76 to the Committee on Fees and the C.P.S. Fee Schedule Committee, to jointly develop premiums, information on number of people to be covered, etc. By approval of the C.M.S.–C.P.S. Liaison Committee report, the Council authorized C.P.S. to study the feasibility of providing new programs with varying income ceilings and fee schedules. The Liaison Committee also recommended disapproval of House of Delegates resolution No. 13 on the basis that the inequities in the A Schedule would be compounded, not eliminated as the House of Delegates resolution suggested.)

(c) The Commission recommended that the Bureau of Research and Planning make a comprehensive study of medical society-sponsored programs in California and information from such study be made available to the membership. On motion duly made and seconded, this recommendation was approved.

15. *Committee on Other Professions:*

Dr. Wayne Pollock, chairman of the Committee on Other Professions, reported on continuing discussions with a committee of the California Osteopathic Association. On motion duly made and seconded, it was voted to approve formation of a joint educational committee of the two associations, to be composed of the deans of the three Southern California medical schools and Dr. Pollock representing the Association and Doctors Bell, Carroll and Grunigen representing the California Osteopathic Association.

On motion duly made and seconded, it was voted to approve a letter to be sent by President Foster to President Dorothy Marsh of the California Osteopathic Association and a press release prepared and to be issued jointly by the two associations. It was also agreed that a letter be sent to the membership, advising them of the history and current status of these discussions.

16. *Committee on Legislation:*

Dr. Kilroy and Mr. Read reported on the results of the 1960 elections and stated that a record number of legislative proposals was anticipated in the 1961 legislative session, many of them emanating from sources which may be inimical to medicine.

17. *Liaison Committee to State Department of Social Welfare:*

Chairman Quinn of the Liaison Committee to the State Department of Social Welfare reported on a meeting held with the department, several phases of which had been covered earlier by Mrs. Evans of the department. On motion duly made and seconded, it was voted to accept this report.

18. *Commission on Public Agencies:*

Chairman Wheeler of the Commission on Public Agencies presented a request from Senator Byrne for nomination of a representative to serve on a Citizens' Advisory Committee on Predators and Rabies. On motion duly made and seconded, it was voted to approve this request, the Committee on Committees to name the representative.

19. *Commission on Community Health Services:*

(a) Chairman MacLaggan discussed proposals which have been given to the Governor for improved medical and environmental care of migratory farm workers. It was agreed that legislative proposals along this line be carefully studied.

(b) A request of the Section on Industrial Health to change its name to Section on Occupational Health was, on motion duly made and seconded, referred to the Committee on Committees for study and recommendation.

(c) A proposed letter to the Joint Commission on Education, urging the maintenance of a program of health education in the schools was approved.

(d) Jack Collins reported on the Governor's Conference on Traffic Safety, in which the Committee on Traffic Safety participated. The committee also recommended that traffic safety committees be established in the county societies, these committees to work with the Department of Motor Vehicles locally and to make recommendations on individual cases referred by that agency. On motion duly made and seconded, it was voted to approve this recommendation and to refer any proposed legislation to the Committee on Legislation.

20. *Liaison Committee to California Hospital Association:*

Dr. MacLaggan reported that the program for hospital-physician relationships would be functioning in four counties next month and that additional hospitals were being contacted to inaugurate the program. He also reported that the Crippled Children's Services of the State Department of Public Health had approved the hospital-physician principles for recognition of hospitals under its program.

21. *Commission on Cancer:*

Report was made on the proposed revision and updating the "Cancer Studies" produced and published by the Commission several years ago. Further report will be made later.

22. *Reorganization of State Government:*

Councilor Anderson called attention to the report of the Committee on Reorganization of the State Government and pointed out that some sections have a bearing on medical practice. It was regularly moved, seconded and voted to request the Committee on Legislation to review such sections and recommend appropriate action.

Recess:

At this point the Council was declared in recess until Sunday, December 11, 1960, at 8:00 a.m.

Reconvention:

The Council was reconvened at 8:00 a.m., Sunday, December 11, 1960, for a general discussion of various problems confronting the medical profession today.

23. *Governor's Committee on Medical Aid and Health:*

Doctors Paul D. Foster and T. Eric Reynolds, members of the Governor's Committee on Medical Aid and Health, reported on the progress of the

committee and on its forthcoming report. It was pointed out that within the next 15 years the state will have need for 1,400 medical graduates annually from its own schools, or approximately three times the present number.

Dr. Francis E. West made a complete report on the functions and operations of the State Board of Medical Examiners. Councilor Quinn, a member of that Board for 12 years, supplemented this report.

On motion duly made and seconded, it was voted to include in the approved report of the ad hoc committee to study the report of the Governor's Committee on Medical Aid and Health a section pointing out that all physicians and the Association are greatly concerned with the need for the licensing authority to exercise some degree of continuing supervision over standards of care.

24. *Foundations for Medical Care:*

Discussion was held on the operations and the position of several foundations for medical care which have been sponsored by county societies in recent years. On motion duly made and seconded, it was voted to refer to the appropriate committee or committees a study of the present and the potentially desirable spheres of activity of various types of underwriters and mechanisms in voluntary health insurance.

25. *Commission on Medical Service—C.P.S. Liaison Committee:*

Discussion was held on the effectiveness of the Commission on Medical Services–C.P.S. Liaison Committee and the question was propounded that this committee might more effectively operate as a direct committee of the Council rather than through a commission. On motion duly made and seconded, it was voted to refer this question to the Committee on Committees for study and report back to the Council at the earliest possible opportunity.

26. *Fees for State Medical Care Programs:*

Discussion was held on possible means of achieving uniform and adequate fees for medical services provided under several programs financed by the State of California. Variations in fee levels in different areas of the state were demonstrated. On motion duly made and seconded, it was voted that state officials be requested to achieve uniform and adequate medical care fees on the basis of allocation of fees by regions through utilization of a composite premium structure under a pooled fund program.

27. *Ad Hoc Committee on History of C.M.A.:*

Councilor Rogers presented a progress report for the ad hoc Committee on History of the C.M.A., pointing out that no author is known to be engaged in preparing such a history at this time but that considerable material is available for such a project. On motion duly made and seconded, it was voted to approve the committee's recommendation that a bibliography of available historical material be sent to each county society, to medical schools and other interested parties.

28. *C.M.A. Mailing List:*

A request for use of the Association's mailing list was presented and, on motion duly made and seconded, was denied.

Adjournment:

There being no further business to come before it, the meeting was adjourned at 3:15 p.m.

SAMUEL R. SHERMAN, M.D., *Chairman*
MATTHEW N. HOSMER, M.D., *Secretary*

In Memoriam

ARKLIN, VIRGIL P. Died in Sun Valley, December 23, 1960, aged 38. Graduate of the University of Southern California School of Medicine, Los Angeles, 1947. Licensed in California in 1947. Doctor Arklin was a member of the Los Angeles County Medical Association.

✚

DAVIES, THOMAS F. Died in 1960, aged 84. Graduate of New York Medical College Flower and Fifth Avenue Hospitals, New York, N. Y., 1898. Licensed in California in 1928. Doctor Davies was a member of the Alameda-Contra Costa Medical Association.

✚

ELLIS, LULA TALBOTT. Died in Duarte, December 27, 1960, aged 94. Graduate of University of Southern California School of Medicine, Los Angeles, 1888. Licensed in California in 1889. Doctor Ellis was a member of the Los Angeles County Medical Association, a life member of the California Medical Association, and a member of the American Medical Association.

✚

FISHMAN, LOUIS ZOLO. Died in Walnut Creek, December 17, 1960, aged 56. Graduate of the University of Illinois College of Medicine, Chicago, 1928. Licensed in California in 1943. Doctor Fishman was a member of the Alameda-Contra Costa Medical Association.

✚

FREEDMAN, EUGENE. Died in Los Angeles, December 26, 1960, aged 63. Graduate of the Universitatea Regele Ferdi-

nand I-lu din Cluj Facultatea de Medicina si Farmacie, Rumania, 1920. Licensed in California in 1945. Doctor Freedman was a member of the Los Angeles County Medical Association.

✤

HILLYER, ERNEST C. Died in Sherman Oaks, December 28, 1960, aged 58. Graduate of the University of Colorado School of Medicine, Denver, 1932. Licensed in California in 1938. Doctor Hillyer was a member of the Los Angeles County Medical Association.

✤

HOLMES, ARTHUR J. Died in Pasadena, December 30, 1960, aged 60. Graduate of Baylor University College of Medicine, Houston, Texas, 1925. Licensed in California in 1927. Doctor Holmes was a member of the Los Angeles County Medical Association.

✤

HORNER, MERLE MEYERS. Died December 17, 1960, aged 48. Graduate of George Washington University School of Medicine, Washington, D. C., 1941. Licensed in California in 1950. Doctor Horner was a member of the Los Angeles County Medical Association.

✤

KING, NORRIS CURTIS. Died in Elsinore, December 29, 1960, aged 66. Graduate of Meharry Medical College, Nashville, Tennessee, 1924. Licensed in California in 1927. Doctor King was a member of the Los Angeles County Medical Association.

✤

LUCAS, WILLIAM PALMER. Died in Oakland, December 16, 1960, aged 80. Graduate of the Western Reserve University School of Medicine, Cleveland, Ohio, 1905. Licensed in California in 1913. Doctor Lucas was a member of the San Francisco Medical Society, a life member of the California Medical Association, and a member of the American Medical Association.

✤

NORTHWAY, WILLIAM HERRICK. Died in Palo Alto, December 23, 1960, aged 57. Graduate of Stanford University School of Medicine, Stanford-San Francisco, 1930. Licensed in California in 1930. Doctor Northway was a member of the Santa Clara County Medical Society.

✤

PETERSON, HERBERT G., JR. Died December 5, 1960, aged 38. Graduate of Harvard Medical School, Boston, Massachusetts, 1948. Licensed in California in 1956. Doctor Peterson was a member of the Los Angeles County Medical Association.

✤

SISSON, CHARLES E. Died in Hollywood, December 26, 1960, aged 87. Graduate of the University of Illinois College of Medicine, Chicago, 1899. Licensed in California in 1915. Doctor Sisson was a retired member of the Los Angeles County Medical Association and the California Medical Association, and an associate member of the American Medical Association.

✤

WEITKAMP, ALFRED H. Died December 14, 1960, aged 78. Graduate of the Denver College of Physicians and Surgeons, Colorado, 1909. Licensed in California in 1916. Doctor Weitkamp was a member of the Los Angeles County Medical Association, a life member of the California Medical Association, and a member of the American Medical Association.

California Medical Association

1961

For complete program see March issue

Annual Session

AMBASSADOR HOTEL • LOS ANGELES

APRIL 30 to MAY 3

- Five Outstanding Guest Speakers
- General Scientific Meetings
- Specialty Scientific Meetings
- Postgraduate Courses
- Medical Motion Picture Symposia
- Technical Exhibits • Scientific Exhibits
- Presidents' Dinner Dance
  Sunday, April 30 • Cocoanut Grove
- House of Delegates
  Opening Session Saturday, 2:00 p.m., April 29
  Sunday, April 30, Tuesday Afternoon, May 2, and Wednesday, May 3
- Registration Daily
  8:30 a.m. to 5:00 p.m. . . . No Registration Fee

PLEASE MAKE HOTEL ROOM RESERVATIONS ONLY THROUGH C.M.A. OFFICE IN SAN FRANCISCO. USE RESERVATION REQUEST FORM ON PAGE 115.

C.M.A. ANNUAL MEETING—1961 SCIENTIFIC SESSIONS

LOCATION	SUNDAY APRIL 30		MONDAY MAY 1		TUESDAY MAY 2		WEDNESDAY MAY 3	
AMBASSADOR HOTEL (Lobby Floor)	A.M.	P.M.	A.M.	P.M.	A.M.	P.M.	A.M.	P.M.
Embassy Room	**9:30 a.m.†** **House of Delegates** †See note below for Opening Session	**2 p.m.** General Meeting Inflammatory Diseases of the Colon	**9 a.m.** General Meeting Diagnostic Methods of Gastroenterology		**9 a.m.** General Meeting Five Guests Five Topics	**4:00 p.m.** **House of Delegates**	**9:30 a.m.** **House of Delegates**	
West Venetian Room	**9 a.m.** Internal Medicine	**2 p.m. & 8 p.m.** Film Symposium	**9 a.m.** Film Symposium	**2 p.m. & 8 p.m.** Film Symposia	**9 a.m.** Film Symposium	**2 p.m. & 8 p.m.** Film Symposia	**9 a.m.** Film Symposium	**2 p.m.** Film Symposium
East Venetian Room	**9 a.m.** Pathology					**2 p.m.** Industrial Medicine Physical Medicine General Practice		**2 p.m.** Public Health
Regency Room	**9 a.m.** Obstetrics and Gynecology	**2 p.m.** Eye Anesthesiology		**2 p.m.** Anesthesiology		**2 p.m.** Obstetrics and Gynecology Anesthesiology	**9 a.m.** Neurology	**2 p.m.** Psychiatry
Grove Lounge	**9 a.m.** Dermatology	**2 p.m.** Ear, Nose, Throat					**9 a.m.** Urology	**2 p.m.** Urology
Lido Room	**9 a.m.** Radiology	**2 p.m.** Radiology			**9 a.m.** Physical Medicine			**2 p.m.** Internal Medicine
Colonial Room	**9 a.m.** General Surgery					**2 p.m.** Disaster Medical Care		
Rose Room	**9 a.m.** Orthopedics							
(Casino Floor) Oval Room A	**9 a.m.** Allergy	**12:30 p.m.** Allergy Luncheon and Business Meeting						**2 p.m.** Pediatrics
WHITE MEMORIAL HOSPITAL	**9 a.m.*** **(Bus at 8 a.m.*)** Postgraduate Course C.M.E.			**2 p.m.** Postgraduate Course C.M.E.		**2 p.m.** Postgraduate Course C.M.E.		
L. A. COUNTY HOSPITAL USC SCHOOL OF MEDICINE (Starts Saturday)	**9 a.m.*** **(Bus at 8 a.m.*)** Postgraduate Course U.S.C.	**2 p.m.** P.G. Course—Cardiac Resuscitation—U.S.C.						

*Buses will leave Ambassador Hotel, Wilshire Entrance.
U.S.C. Courses will start on Saturday.
†**Opening meeting, House of Delegates, 3:00 p.m., Saturday, April 29.**

TECHNICAL EXHIBITS—Sunset Room, Ballroom and Boulevard Room, Casino Floor.
SCIENTIFIC EXHIBITS—Venetian Room Foyer.
COUNCIL OF THE C.M.A. MEETS DAILY AT 7:30 A.M. IN THE FRENCHETTE ROOM.

4 POSTGRADUATE COURSES

C.M.A. ANNUAL SESSION • *April 29–May 3, 1961* • *Los Angeles*

THE CALIFORNIA MEDICAL ASSOCIATION *in cooperation with the Medical Schools of* UNIVERSITY OF SOUTHERN CALIFORNIA *and* COLLEGE OF MEDICAL EVANGELISTS, *will present four Postgraduate Courses at the time of the Annual Session in April. These courses will be clinically oriented and will include case presentations.*

Choose the course which most interests you, follow the course, and the 1961 session will send you back to your practice stimulated and refreshed.

Look for the program giving complete details which will arrive in your office in March.

• By COLLEGE OF MEDICAL EVANGELISTS

Clinical Neurology—9 hours

Time: Sunday, April 30, 9:00 a.m. to 12:00 noon; Monday and Tuesday, May 1 and 2, 2:00 p.m. to 5:00 p.m.

Place: White Memorial Hospital. *Fee:* $25.00

• By UNIVERSITY OF SOUTHERN CALIFORNIA

Uses and Limitations of Laboratory Tests—9 hours

Time: Saturday, April 29, 9:00 a.m. to 12:00 noon; 1:30 p.m. to 5:00 p.m.; Sunday, April 30, 9:00 a.m. to 12:00 noon.

Place: Los Angeles County Hospital. *Fee:* $25.00

Practical Gynecology—9 hours

Time: Saturday, April 29, 9:00 a.m. to 12:00 noon; 1:30 p.m. to 5:00 p.m.; Sunday, April 30, 9:00 a.m. to 12:00 noon.

Place: USC School of Medicine. *Fee:* $25.00

Cardiac Resuscitation—2 hours

SECTION I*—Saturday, April 29, 9:00 a.m. to 11:00 a.m.

SECTION II*—Sunday, April 30, 2:00 p.m. to 4:00 p.m.

Place: USC School of Medicine. *Fee:* $30.00

*Each Section is all-inclusive. Each Section is limited to 15 registrants from areas other than San Francisco or Los Angeles.

APPLICATION FOR ENROLLMENT

Mail to: POSTGRADUATE ACTIVITIES, CALIFORNIA MEDICAL ASSOCIATION
2975 Wilshire Boulevard, Los Angeles 5, California

With check or money order in the amount of $_____ made payable to *California Medical Association*

Name_____

Address_____

I am in General Practice_____ I limit my practice to_____

Medical School Attended_____ Year of Graduation_____

Please enroll me in the course indicated by ✓.

☐ 1. **Clinical Neurology** (9-hour course, Sunday morning; Monday and Tuesday afternoons)—$25.00

☐ 2. **Uses and Limitations of Laboratory Tests** (9-hour course, Saturday, all day; Sunday morning)—$25.00

☐ 3. **Office Gynecology** (9-hour course, Saturday, all day; Sunday morning)—$25.00

☐ 4a. **Cardiac Resuscitation—Section I**—Saturday morning—$30.00

☐ 4b. **Cardiac Resuscitation—Section II**—Sunday afternoon—$30.00

APPLICATION FOR HOUSING ACCOMMODATIONS

FOR YOUR CONVENIENCE in making hotel reservations for the coming meeting of the **California Medical Association,** April 30*-May 3, 1961, Los Angeles, hotels and their rates are at the right. Use the form at the bottom of this page, indicating your first and second choice. Because of the limited number of single rooms available, your chance of securing accommodations of your choice will be better if your request calls for rooms to be occupied by two or more persons. **All requests for reservations must give definite date and hour of arrival as well as definite date and approximate hour of departure; also names and addresses of all occupants of hotel rooms must be included.**

Ninetieth Annual Session
CALIFORNIA MEDICAL ASSOCIATION
Los Angeles, California
APRIL 30*-MAY 3, 1961

HOTEL ROOM RATES†

	Single	Twin Beds	Suites
AMBASSADOR HOTEL			
3400 Wilshire Boulevard			
Main Building	14.00-24.00	18.00-28.00	40.00-58.00
Garden Studios	22.00-34.00	24.00-36.00	54.00-66.00
CHAPMAN PARK HOTEL			
3405 Wilshire Boulevard	10.00-11.00	14.00-16.00	20.00-28.00
Bungalows		18.00	25.00-28.00
THE GAYLORD HOTEL			
3355 Wilshire Boulevard	9.00-10.00	12.00-15.00	Single: 25.00 Double: 35.00
HOTEL CHANCELLOR			
3191 West Seventh Street	8.00-10.00	12.00	
SHERATON-WEST			
(formerly Sheraton-Town House)			
2961 Wilshire Boulevard	12.50-20.00	17.50	34.00

ALL RESERVATIONS MUST BE RECEIVED BEFORE: APRIL 1, 1961

***April 29: House of Delegates will start with afternoon meeting Saturday, April 29.**

†The above quoted rates are existing rates but are subject to any change which may be made in the future.

CALIFORNIA MEDICAL ASSOCIATION—Dept. 74
693 Sutter Street
San Francisco 2, California

Please reserve the following accommodations for the 90th Annual Session of the California Medical Association, in Los Angeles April 30-May 3, 1961. (House of Delegates members: First meeting of House begins Saturday afternoon, April 29.)

Single Room $..... Twin-Bedded Room $.....

Small Suite $..... Large Suite $..... Other Type of Room $.....

First Choice Hotel..... Second Choice Hotel.....

ARRIVING AT HOTEL (date):..... Hour:..... A.M. ..... P.M. } Hotel reservations will be held until

Leaving (date) ..... Hour:..... A.M. ..... P.M. } 6:00 p.m., unless otherwise notified

THE NAME OF EACH HOTEL GUEST MUST BE LISTED. Therefore, please include the names of both persons for each twin-bedded room requested. Names and addresses of all persons for whom you are requesting reservations and who will occupy the rooms asked for:

.....

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.....

.....

Individual Requesting Reservations—Please print or type Officer?..... Delegate?..... Alternate?.....

Name..... County.....

Address..... City and State.....

11th ANNUAL

REGIONAL POSTGRADUATE INSTITUTE

WEST COAST COUNTIES

Presented by Committee on Postgraduate Activities of the California Medical Association, in cooperation with Monterey, Santa Cruz and San Benito Counties Medical Societies, and the College of Medical Evangelists.

Guest Speaker: Owen H. Wangensteen, M.D., Professor and Chief, Department of Surgery, University of Minnesota Medical School; President, American College of Surgeons, 1959-60.

Del Monte Lodge, Pebble Beach

March 2 and 3, 1961

PROGRAM

THURSDAY, MARCH 2

Morning Session

PROBLEMS OF THE SENIOR CITIZEN

9:30-10:00—**Urological Problems Involved in Geriatric Practice**—Roger W. Barnes, M.D.

10:00-10:30—**Recognition and Management of Cerebral Vascular Accidents**—Julius Bauer, M.D.

10:30-11:30—**Cancer of the Alimentary Tract of the Aged**—Owen H. Wangensteen, M.D.

11:45-12:30—**Geriatric Panel:** Edward R. Bloomquist, M.D., Moderator; Roger W. Barnes, M.D.; Julius Bauer, M.D.; Owen H. Wangensteen, M.D.

Afternoon Session

ENDOCRINOLOGY

2:00-2:40—**Pituitary Adrenal Axis**—Julius Bauer, M.D.

2:40-3:20—**The Use of Progestins in Practice**—Gordon P. Griggs, M.D.

3:20-4:00—**Pediatrics**—Robert F. Chinnock, M.D.

4:15-5:00—**Endocrinology Panel:** C. Joan Coggin, M.D., Moderator; Julius Bauer, M.D.; Gordon P. Griggs, M.D.; Robert F. Chinnock, M.D.

7:30—**Banquet** (wives invited)—Speaker of national repute, followed by informal dancing.

FRIDAY, MARCH 3

Morning Session

GENERAL SURGERY

9:30-10:00—**Recognition of Cardiac Arrest**—Edward R. Bloomquist, M.D.

10:00-10:30—**Recognition and Management of Endometriosis**—Gordon P. Griggs, M.D.

10:30-11:30—**Surgical Facets of the Peptic Ulcer Problem**—Owen H. Wangensteen, M.D.

11:45-12:30—**General Surgery Panel:** Albert C. Daniels, M.D., Moderator; Edward R. Bloomquist, M.D.; Gordon P. Griggs, M.D.; Owen H. Wangensteen, M.D.

Afternoon Session

PROBLEMS OF THE JUNIOR CITIZEN

2:00-2:40—**Problems in Adolescence**—Robert F. Chinnock, M.D.

2:40-3:20—**Pediatric Urology**—Roger W. Barnes, M.D.

3:20-4:00—**Selection of Patients for Cardiac Surgery**—C. Joan Coggin, M.D.

4:15-5:00—**Pediatric Panel:** Julius Bauer, M.D., Moderator; Robert F. Chinnock, M.D.; Roger W. Barnes, M.D.; C. Joan Coggin, M.D.

HOST: Monterey County Medical Society . . . **REGIONAL CHAIRMAN:** A. F. Kandlbinder, M.D., 835 Cass Street, Monterey, California . . . **INSTITUTE FEE:** $15.00. For additional information, contact Postgraduate Activities Office, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5. **All California Medical Association members and their families are cordially invited to attend.**

11th ANNUAL
REGIONAL POSTGRADUATE INSTITUTE
NORTH COAST COUNTIES

MIRRORS OF SYSTEMIC DISEASE—Presented cooperatively by University of California School of Medicine, San Francisco, North Coast Counties Medical Societies, and California Medical Association Committee on Postgraduate Activities.

Flamingo Hotel, **4th Street and Farmers Lane**

SANTA ROSA, MARCH 23-24, 1961

• Certain key parts of the routine examination of patients act as the alert for the existence of generalized systemic disease. Whatever kind of practice a physician is engaged in, these remain among the most important parts of his examination. This Institute is devoted to some of these aspects. The problems of further investigation and treatment of patients with these manifestations will be discussed in an interdisciplinary manner. In the panels, patient demonstrations and discussion groups, there will be full opportunity for active participation by the registrants.

Guest Speaker: **WALTER C. LOBITZ, JR., M.D.,** Professor of Dermatology and Head of the Division of Dermatology, University of Oregon Medical School, Portland, Oregon.

PROGRAM

THURSDAY, MARCH 23

Morning Session

PERIPHERAL BLOOD—A MIRROR OF SYSTEMIC DISEASE

9:00-10:00—**Lecture**—Paul M. Aggeler, M.D.

10:15-12:00—**Panel Discussion.** Moderator: Paul M. Aggeler, M.D.; Panel: William C. Deamer, M.D.; Richard E. Gardner, M.D.; Wallace V. Epstein, M.D.; Glenn E. Sheline, M.D.

Afternoon Session

THE SKIN—A MIRROR OF SYSTEMIC DISEASE

1:30-2:30—**Lecture**—Walter C. Lobitz, Jr., M.D.

2:45-4:45—**Panel Discussions and Presentation of Patients** (Panels meet simultaneously—and will rotate). Registrants to remain in same room throughout.

PANEL 1: **Presentation of Four Patients Accenting Collagen and Related Diseases**—Wallace V. Epstein, M.D.; William Whiting, M.D.; E. Manfred Bloner, M.D.

PANEL 2: **Presentation of Four Patients Accenting Neoplastic Disease**—John Pennington, M.D., Robert Butler, M.D.; Glenn E. Sheline, M.D., R. L. Zieber, M.D.

PANEL 3: **Presentation of Four Patients Accenting Peripheral Vascular Disease**—Richard E. Gardner, M.D.; Bradford Lundborg, M.D.; A. A. Tratar, M.D.

PANEL 4: **Presentation of Four Patients Accenting Infections**—William C. Deamer, M.D.; Walter Weber, M.D.; George Rostel, M.D.

(Lecturer will rotate from one panel to another and some patients will be presented to more than one panel.)

7:30—**Institute Banquet—Informal Dancing.**

FRIDAY, MARCH 24

Morning Session

THE URINE—A MIRROR OF SYSTEMIC DISEASE

9:00-10:00—**Lecture**—Max Rukes, M.D.

10:15-12:00—**Panel Discussion.** Moderator: Max Rukes, M.D. Panel: Vincent Di Raimondo, M.D.; Walter C. Lobitz, Jr., M.D.; Edward C. Hill, M.D.; Milton L. Rosenberg, M.D.; Felix O. Kolb, M.D.

Afternoon Session

THE EYE—A MIRROR OF SYSTEMIC DISEASE

1:30-2:30—**Lecture**—William F. Hoyt, M.D.

2:45-4:45—**Panel Discussions and Presentation of Patients** (Panels meet simultaneously—and will rotate). Registrants to remain in same room.

PANEL 1: **Presentation of Four Patients Showing Complications of Pregnancy and Hypertension**—Edward C. Hill, M.D.; Robert Huntington, M.D.; Charles Kelly, M.D.

PANEL 2: **Presentation of Four Patients Showing Endocrine Problems and Pituitary Problems**—Norman Panting, M.D.; Daniel McCaskill, M.D.; Felix O. Kolb, M.D.

PANEL 3: **Presentation of Four Patients with Urological Problems**—John Bodle, M.D.; Stanford Hampton, M.D.; Milton L. Rosenberg, M.D.

PANEL 4: **Presentation of Four Diabetic Patients**—Vincent Di Raimondo, M.D.; Vernon Lightfoot, M.D.; John H. Wuest, Jr., M.D.

(Lecturer will rotate from one panel to another and some patients will be presented to more than one panel.)

HOST: Sonoma County Medical Society . . . **REGIONAL CHAIRMAN:** Milton A. Antipa, M.D., 50 Montgomery Drive, Santa Rosa. **INSTITUTE FEE:** $15.00. For additional information contact Postgraduate Activities office, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5. **All California Medical Association members and their families are cordially invited to attend.**

PUBLIC HEALTH REPORT

MALCOLM H. MERRILL, *M.D., M.P.H.*
Director, State Department of Public Health

A "QUARTER-WAY" house program, a unique rehabilitative venture for alcoholics, will be started soon in San Francisco for men jailed for drinking offenses.

The program, supported by state funds, and carried out by the Adult Guidance Center and the Northern California Service League, will assist about 50 men released each month from jail who ask for further assistance with their drinking problems and personal rehabilitation.

More than 300 alcoholic men are discharged each month from San Francisco County Jail No. 2. In the alcoholic rehabilitation clinic in the San Bruno jail a few are considered fair candidates for rehabilitation.

Details are still being worked out but this is generally how the program will operate:

Dr. Thomas G. Bond, a psychiatrist, and psychiatric social workers assigned to the program from the Guidance Center, will select about 50 men each month who ask for further assistance. After release from jail, these selected will be housed in a "quarter-way" house some distance from Skid Row and near downtown employment centers.

The Service League, a privately-financed social organization devoted to helping released jail and penitentiary inmates find employment and achieve a measure of social reintegration, will use the state funds to provide board and room for periods up to two weeks. Housing and meals will cost $25 per man, and up to $5 per week will be available for cleaning, laundry, shaving materials and transportation to job locations.

The "quarter-way" program implies that alcoholics who want help will be assisted on the path to personal rehabilitation but will not be supported during the entire reentry into society.

The Guidance Center plans follow-up counseling and casework with the house residents and will work with other city agencies in attempting to find employment for them. A psychiatric social worker will be at the house half-time daily for personal consultation but the primary focus will be on obtaining employment.

* * *

A three-year research grant to determine the optimum levels of fluoride ion concentration in California water supplies for different temperature zones has been awarded the Division of Dental Health by the National Advisory Dental Health Research Council.

Temperature is directly related to the fluid intake of individuals. To achieve a given intake of fluoride ion in different temperature zones, the concentration of the fluoride ion should vary inversely with the temperature. An excess of fluorides will result in the mottling of teeth, while an inadequate supply will not provide the optimum resistance to caries.

The project is designed to study the dental experience of children in varying fluoride and temperature zones to provide more exact information to establish what the optimum desirable levels are.

* * *

The year 1960 closed with 365 cases of paralytic poliomyelitis reported, compared with 418 for the previous year. Deaths numbered 23, four more than recorded in 1959.

The pattern of the disease has remained unchanged during recent years. About 80 per cent of the cases occurred in non-vaccinated or poorly vaccinated persons, some 40 per cent occurring in children below the age of five years.

It is not anticipated that oral poliomyelitis vaccine will be available before the end of 1961 in any appreciable quantity, so vaccination with Salk vaccine should be continued.

National Fall Conference

IN KEEPING with the concept that "One's judgment can never be better than one's information," I recently reviewed the Fall Conference of State Auxiliaries that was held in Chicago, October 2-5. I would like to present to you a few of the highlights of that stimulating meeting.

The formal opening of the conference was preceded by a dinner at which state auxiliary presidents and presidents-elect sat in groups organized according to geographic boundaries. The Western Division, which included California, was presided over by Mrs. Hiram D. Cochran, who had cleverly decorated the tables with dolls and maps showing resources, industry and entertainment representative of each state. Membership, and particularly membership-at-large, which every state is anxious to include, was discussed. It was of real interest to note that many state and county medical societies pay their auxiliary's dues. Payment of auxiliary dues by the state or county medical society stimulates and encourages participation in auxiliary activities by the wives. (Men—please note.)

On Monday morning the conference was opened formally. Dr. F. J. Blasingame, executive vice-president of the American Medical Association, alerted us to the trying times facing medicine and asked us to dedicate ourselves to further participation in American life. He stressed the importance of auxiliary members' being informed on the important issues of the day. He emphasized that as doctors' wives and as citizens of our communities we should know why we do not believe in socialized medicine, and be keenly aware of its inferior patient care and more expensive operation.

Dr. Edward Annis, of Florida, spoke on the role of the physician's wife in legislation. He urged us to not only be cognizant of medical matters, but also to be informed on such subjects as minimum wages and industrial and labor problems. Being well informed, he stressed, permits the wives to authoritatively relay information to others with whom they talk. He was insistent that we vote for the party which would keep the practice of medicine free. There are already too many governmental controls, he said, and self-respect must supersede security.

A bridge table skit demonstrated the various phases of the life of a physician's wife—her part in politics; her role as a public relations person in her community (i.e., when she meets her husband's patients); her telephone conversations with patients; her attitude toward other physicians' wives, and her behavior when new in the community.

A clever skit, "The Public Image of a Doctor's Wife," was presented in a public opinion research interview. This depicted how the average person sees a physician's wife as a respectable, kind and helpful citizen. The civic leader sees her as a capable woman in community and church affairs. The newspaperwoman takes note of the work of the entire auxiliary —the one million dollars in scholarships to nurses since 1950, the contributions to medical schools, and the like.

Safety was stressed, with emphasis on SWAT, (Safety Water Activity Training). Every physician's child should learn to swim. We were informed that drowning is the fourth leading type of fatal accident, and we learned that each year many children between 1 and 2 years of age swallow lethal poison. The National Auxiliary had two sets of slides on poison control. Traffic safety, too, was emphasized. Some states require health certificates to obtain a driver's license. It was interesting to learn that one out of ten automobile accidents involves a teen-aged driver.

We were informed by Dr. George Lull, president of American Medical Education Foundation, that the Auxiliary gift to the fund had increased from its initial $10,000 in 1950 to $175,000 in 1959.

The membership presentation stressed the fact that collective efforts bring greater results than individual efforts. Therefore, the greater our membership, the greater is our strength. Enthusiasm and friendliness are essential.

Robert M. Hanson, of the Office of Civil Defense and Mobilization in Michigan, discussed Shelters for Survival. It was interesting to learn from his message that USSR has trained 187 million Russians in civil defense; that they are building shelters and that the Communists have the upper hand in the "balance

of terror." Our own Civil Defense has readied 2,000 emergency hospitals of 200 beds each. A home warning system called NEAR is in effect. People must save themselves—no agency can save people. He went on to say that it is a sign of realization of the times when we build ourselves shelters instead of swimming pools.

Dr. Leonard Larson, president-elect of the A.M.A., praised the Auxiliary for the 3 million hours of volunteer services that have been given through the Auxiliary and other organizations, and for our work for Legislation, for A.M.E.F., and for other important committees.

On Wednesday morning, after touring the beautiful A.M.A. headquarters, we were addressed by Dr. Ernest B. Howard, assistant executive vice-president of the A.M.A. He enlightened us on the first National Congress on Mental Health, which will be sponsored by the A.M.A.; the scholarships and loans available for medical students, probably through A.M.E.F.; and on the commission which has been appointed to study all facets of the cost of medical care. He congratulated the auxiliaries on helping defeat the Forand bill.

And so ended our conference. I am sure that the consensus of those who attended this exhilarating meeting was that we, as an auxiliary, are definitely needed; that we are respected for the help we have given and can give. This was most obvious from the tone of every speaker.

MRS. SAMUEL GENDEL
President, Woman's Auxiliary to the California Medical Association

NEWS & NOTES

NATIONAL · STATE · COUNTY

LOS ANGELES

Dr. Leo G. Rigler, Los Angeles, was recently presented with the gold medal of the American College of Radiology "for distinguished and extraordinary service to the American College of Radiology and the profession for which it stands."

* * *

Officers for 1961 for the **Metropolitan Dermatological Society** of Los Angeles, elected at the annual meeting, are as follows: President, Dr. Murray C. Zimmerman, Whittier; vice-president, Dr. Rose B. Saperstein, Los Angeles; secretary-treasurer, Dr. John W. Carney, Los Angeles; program chairman, Dr. Sidney J. Rose, Los Angeles.

SAN FRANCISCO

Some 500 physicians and attorneys from the Western states are expected to attend a meeting on **problems in the medicolegal field** at the Mark Hopkins Hotel, March 10 and 11.

Sponsored by the American Medical Association's Legal and Socio-Economic Division, the meeting will have panel discussions on Res Ipsa Loquitur in Medical Professional Liability Cases, Use and Misuse of Demonstrative Evidence in Personal Injury Cases, Expert Medical Testimony, and the Status of Physician-Attorney Relations.

A half-day session will be devoted to **developments and trends in judicial procedures.** Included will be a consideration of the separation of the questions of liability and damages in the trial of a case, impartial medical testimony projects, developments in pre-trial areas and discovery procedures, and the "liability without fault" approach in all personal injury litigation.

Another half-day session will deal with a discussion of the most important cases in the medicolegal field during the past 18 months. The cases will not only be discussed by legal experts from the point of view of the plaintiff and the defendant, but also by physicians as to the medical implications of the decisions rendered.

Registration fee for the conference will be $5 to cover the cost of a luncheon on Saturday and a copy of the proceedings. Advance registration cards may be obtained by writing: C. Joseph Stetler, director, Legal and Socio-Economic Division, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois.

This is the first in a series of three medico-legal meetings being sponsored by the A.M.A. this year. Other conferences will be held in Louisville, April 14 and 15, and in New York, April 28 and 29.

* * *

The Western Regional Meeting of the **American College of Gastroenterology** will be held in San Francisco, Sunday, March 5, 1961. The sessions, which will be held at the Fairmont Hotel, will commence at 9:00 a.m.

The western region consists of the states of Arizona, California, Colorado, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington and Wyoming.

Members of the medical profession are invited to attend. A copy of the program may be obtained from the Secretary, American College of Gastroenterology, 33 West 60th Street, New York 23, N. Y.

Dr. Julius H. Comroe, Jr., director of the Cardiovascular Research Institute, University of California Medical Center, San Francisco, was a recipient last month of a Distinguished Achievement Award from *Modern Medicine,* a medical journal published in Minneapolis. Dr. Comroe was honored for "leadership in investigation of pulmonary function and clinical use of autonomic drugs."

Ten recipients are selected each year by the editorial board of *Modern Medicine* from nominations made by deans of medical schools, leaders of medical organizations and readers of the journal.

* * *

The second of the yearly **Low-Beer Memorial Lectures** will be held at the University of California Medical Center, San Francisco, on March 2, 1961. **Dr. Jean Bouchard,** associate professor of radiology, Faculty of Medicine, McGill University, and radiologist, Royal Victoria Hospital, Montreal, Canada, will speak on "Radiation Therapy in the Management of Pituitary Tumours." The lecture will be held in the Auditorium, Medical Sciences Building, at 8 p.m.

The Low-Beer Memorial Lectureship in Radiation Therapy was established in honor of the late Dr. Bertram V. A. Low-Beer, professor of radiology at the University until his death in 1955.

In addition to the formal lecture, Dr. Bouchard will participate in five conferences to be held in the Medical Sciences Building and Moffitt Hospital February 28 and March 1, 2 and 3, to which the members of the medical profession are invited.

Further information may be obtained from Dr. Franz Buschke, professor of radiology, UC Medical Center, San Francisco 22.

SAN JOAQUIN

Dr. William Brock of Stockton became president of the San Joaquin County Medical Society at the annual meeting in January, succeeding Dr. C. A. Lucky. Dr. John T. McNally was elected vice-president and Dr. Dora Ames Lee was reelected secretary-treasurer.

SANTA CLARA

The Fourth Annual Palo Alto Medical Clinic Symposium, sponsored by the clinic and the Palo Alto Medical Research Foundation, will be presented Saturday, April 15, 1961. Further information may be obtained from Dr. John F. Weigen, program chairman, Palo Alto Medical Clinic, 300 Homer Avenue, Palo Alto.

SOLANO

Dr. Carlton C. Purviance, Vallejo, was installed as president of the Solano County Medical Society at a meeting of the organization in January. Dr. William J. Olson, Fairfield, was elected president-elect and Dr. William S. Hebert, Vallejo, secretary-treasurer.

GENERAL

Two Southern California physicians were reappointed to the **Medical Care Advisory Committee** of the State Social Welfare Board late in January. They are Dr. William F. Quinn, Los Angeles, and Dr. Edward Lee Russell, Health Officer of Orange County.

The medical care committee advises the Welfare Board on matters related to the state's Medical Care program. Both Dr. Quinn and Dr. Russell have served on the committee since 1957, when the Medical Care program was initiated.

* * *

The forty-second annual session of the **American College of Physicians** will be held at Miami Beach, Florida, May 8 to 12.

POSTGRADUATE
EDUCATION NOTICES

THIS BULLETIN of the dates of postgraduate education programs and the meetings of various medical organizations in California is supplied by the Committee on Postgraduate Activities of the California Medical Association. In order that they may be listed here, please send communications relating to your future medical or surgical programs to Postgraduate Activities, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5.

UNIVERSITY OF CALIFORNIA AT LOS ANGELES

Clinical Traineeships — Anesthesia, Dermatology and Pediatric Cardiology. Dates by arrangement. Minimum period—two weeks. Fee: Two weeks, $150.00; four weeks, $250.00.

Management of Fractures and Dislocation, and Application of Casts, Splints and Bandages. Thursday through Saturday, February 23-25. Eighteen hours. Fee: Lecture and Lab $70.00; Lecture only $40.00.

Use of Indirect Ophthalmoscopy in Retinal Detachment Surgery. Thursday through Saturday, March 2-4. Eighteen hours.*

Psychiatry in Medicine. Friday and Saturday, March 10 and 11. Twelve hours. Fee: $2.50.

Management of Pain by Therapeutic Nerve Blocks —Harbor Hospital. Friday through Sunday, April 7 through 9. Eighteen hours. Fee: $55.00 (includes two luncheons).

Israel—Clinical Postgraduate Program (sessions to be held in Jerusalem and Tel Aviv). April 20 through 28. Thirty-two hours. Fee: $200.00.

Management of Trauma—Harbor Hospital. Friday and Saturday, May 19 and 20. Nine hours.*

Gerontology. Friday and Saturday, May 19 and 20. Twelve hours.*

Common Emergencies in Clinical Practice. Friday and Saturday, May 26 and 27. Twelve hours. Fee: $40.00.

Dermatology in Clinical Practice. Tuesday, July 11. Six hours. Fee: $20.00.

Advanced Seminars in Dermatology (for Dermatologists). Wednesday through Sunday, July 12 through 16. University Conference Center, Lake Arrowhead. Fourteen and one-half hours. Fee: $150.00 (includes room and meals).

Infertility. Friday and Saturday, July 14 and 15.†

Advanced Seminar on Infertility. Sunday through Wednesday, July 16 through 19. University Conference Center, Lake Arrowhead.†

General Pediatrics. Wednesday through Sunday, August 2 through 6. University Conference Center, Lake Arrowhead. Sixteen hours. Fee: $150.00 (includes room and meals).

Endocrinology. Friday and Saturday, August 4 and 5.†

For information on courses for physicians or ancillary personnel *contact:* Thomas H. Sternberg, M.D., assistant dean for Continuing Medical Education, U.C.L.A. Medical Center, Los Angeles 24. BRadshaw 2-8911, Ext. 7114.

*Fee to be announced.
†Hours and fees to be announced.

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

Medicine for General Practitioners. Mount Zion Hospital. Monday through Friday, February 20 through 24. Thirty-five hours. Fee: $85.00.

Diseases of the Nervous System in Childhood. Thursday through Saturday, March 2 through 4. Twenty-one hours. Fee: $50.00.

Urology for Non-Urologists. Friday and Saturday, March 10 and 11. Fourteen hours.*

Perinatal Problems, Children's Hospital. Saturday, March 11. Seven hours. Fee: $12.50.

Diagnostic Radiology. Wednesday through Sunday, March 15 through 19. Thirty-five hours.*

Evening Lecture Series in Medicine, Eden Hospital. Tuesday evenings, April 4 through May 23.†

Laboratory Investigation of Endocrine Disorders. Friday through Sunday, April 7 through 9. Twenty-one hours.*

General Surgery. Thursday and Friday, April 20 and 21. Fourteen hours.*

Ear-Nose-Throat. Thursday through Saturday, May 11 through 13. Twenty-one hours.*

Proctology. Thursday and Friday, May 18 and 19. Fourteen hours.*

Water, Salts and Steroids. Thursday through Saturday, May 25-27. Twenty-one hours.*

A Course in Ophthalmology. Thursday through Saturday, June 1-3. Twenty-one hours.*

Electrocardiography. Friday and Saturday, June 9 and 10. Fourteen hours.*

A Course in Psychiatry for Physicians in General Practice. Thursday through Saturday, June 15-17. Twenty-one hours.*

Cerebral Palsy. Thursday and Friday, June 29 and 30. Fourteen hours.*

Fundamental Practices of Radioactivity and the Diagnostic and Therapeutic Uses of Radioisotopes. Two or three month course limited to one enrollee per month. Fee: $350.00.

For information on courses for physicians or ancillary personnel *contact:* Seymour M. Farber, M.D., assistant dean, Department of Continuing Medical Education, University of California Medical Center, San Francisco 22. MOntrose 4-3600, Ext. 665.

PRESBYTERIAN MEDICAL CENTER, SAN FRANCISCO

Diabetes and Thyroid Disease: Current Methods in Diagnosis and Treatment. Saturday, February 25. Eight hours. Fee: $25.00.

The Four R's of Fractures: Recognition, Reduction, Retention, Rehabilitation. Saturday, March 11. Eight hours. Fee: $25.00.

Problems in Therapy of Cardiac Disease. Sunday, April 9. Eight hours. Fee: $25.00.

Problems in Neurology and Neurosurgery. Saturday, May 6. Eight hours. Fee: $25.00.

Psychological Problems in General Practice. Sunday, May 21. Eight hours. Fee: $25.00.

Horizons in Surgery. Saturday, June 17. Eight hours. Fee: $25.00.

Note:
Each one of the conferences listed above..... $ 25.00
A series of any 5 conferences..... 100.00

Operable Heart Disease. Friday and Saturday, March 3 and 4.

Conference on Keratoplasty. Wednesday through Friday, March 8 through 10. Limited enrollment.

General Review Course for Practicing Physicians. Thursday through Saturday. March 16 through 18.

Conference on Strabismus. Wednesday through Friday. July 12 through 14.

Contact: Arthur Selzer, M.D., program committee chairman, Presbyterian Medical Center, Clay and Webster Sts., San Francisco 15, WEst 1-8000, Ext. 303.

UNIVERSITY OF SOUTHERN CALIFORNIA, LOS ANGELES

Nuclear Medicine:

Part II, eight weeks. Fee: $350.00.
Part III, twelve weeks. Fee: $350.00.

Ward Walks in Rare Diseases. Thursday evenings, May 4 to July 6, 7:30-9:30 p.m. Los Angeles County Hospital. Tuition: $100.00.

Hawaii Course. August 2 through 18. The USC School of Medicine will offer the 4th Postgraduate Refresher Course to be held in Honolulu and on board the S.S. Matsonia. (As a time and money saver, air travel is also possible.)

Cardiac Resuscitation. Each Wednesday by appointment, 4 to 6 p.m. USC Medical Research Building, Room 211, 2025 Zonal Avenue. Tuition: $30.00. (Each session all-inclusive.)

Basic Home Course in Electrocardiography. One year postgraduate series, electrocardiogram interpretation by mail. Physicians may register at any time and receive all 52 issues. Fifty-two weeks. Fee: $100.00.

Advance Home Course in Electrocardiography. One year postgraduate series, electrocardiogram interpretation by mail. Fifty-two issues: $85.00. Physicians may register at any time.

Contact: Phil R. Manning, M.D., Associate Dean and Director, Postgraduate Division, University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 33. CApital 5-1511.

COLLEGE OF MEDICAL EVANGELISTS

SURGICAL ANATOMY (Dissection, Lectures and Demonstrations):

Head and Neck. Monday and Wednesday, April 19 through May 31. Sixty-three hours. Fee: $75.00.

SURGICAL ANATOMY (Lectures and Demonstrations only):

Head and Neck. Wednesdays, April 12 through May 31. Twenty-four hours. Fee: $35.00.

Alumni Postgraduate Convention Refresher Courses, March 12 and 13, on the campus of the College of Medical Evangelists at White Memorial Hospital.

Joint Manipulation. Monday through Friday, March 20 through 24. Twenty hours. Fee: $100.00.

Tropical Public Health. Monday through Friday, April 3 through 28. Fee: $65.00.

Clinical Traineeships available in clinical departments by arrangement with Postgraduate Division and Postgraduate Chairman of department involved. In addition to those listed other traineeships in other departments can be arranged. Eighty hours minimum. Limited enrollment. Begin when individually arranged.

1. **Anesthesia.** Six months. 250 to 300 hours. Fee: $350.00.
2. **Internal Medicine.** Two weeks to nine months.
3. **Pulmonary Diseases** (can be arranged).
4. **Traumatology.** One month. 160 hours. Fee: $125.00.
5. **Urology** (can be arranged).

For information contact: Division of Postgraduate Medicine, College of Medical Evangelists, 1720 Brooklyn Ave., Los Angeles 33. ANgelus 9-7241, Ext. 214.

CALIFORNIA MEDICAL ASSOCIATION ANNUAL SESSION POSTGRADUATE COURSES

Clinical Neurology in cooperation with College of Medical Evangelists. White Memorial Hospital. Sunday, April 30, 9-12 a.m.; Monday and Tuesday, May 1 and 2, 2-5 p.m. Fee: $25.00.*

Office Gynecology in cooperation with USC School of Medicine. Los Angeles County Hospital. Saturday, April 29, 9-12 a.m. and 2-5 p.m.; Sunday, April 30, 9-12 a.m. Fee: $25.00.*

Use and Limitations of Laboratory Tests in cooperation with USC School of Medicine. Los Angeles County Hospital. Saturday, April 29, 9-12 a.m. and 2-5 p.m.; Sunday, April 30, 9-12 a.m. Fee: $25.00.*

Cardiac Resuscitation: in cooperation with USC School of Medicine. USC Research Building, Room 211, 2025 Zonal Avenue, Los Angeles 33. Section I: Saturday, April 29, 2-4 p.m.; Section II: Sunday, April 30, 2-4 p.m. (each Section is all-inclusive). Each Section is limited to 10 registrants from areas other than Los Angeles and San Francisco. Fee: $30.00, each Section.*

CALIFORNIA MEDICAL ASSOCIATION POSTGRADUATE CIRCUIT COURSES

For Dunsmuir, Redding, Chico and Marysville in cooperation with Stanford University School of Medicine. Begins week of March 5, 1961.

For Eureka, Ukiah, Napa and Auburn in cooperation with University of California School of Medicine, San Francisco. Begins week of March 5, 1961. *Napa* course begins March 15, 1961.

CALIFORNIA MEDICAL ASSOCIATION POSTGRADUATE INSTITUTES—1961

West Coast Counties, March 2 and 3, Del Monte Lodge, Pebble Beach, in cooperation with College of Medical Evangelists. *Chairman:* A. F. Kandlbinder, M.D., 835 Cass Street, Monterey.

North Coast Counties, March 23 and 24, Flamingo Hotel, Santa Rosa, in cooperation with University of California, San Francisco. *Chairman:* Milton A. Antipa, M.D., 50 Montgomery Drive, Santa Rosa.

San Joaquin Valley, April 14 and 15, Ahwahnee Hotel, Yosemite, in cooperation with UCLA School of Medicine. *Chairman:* J. Malcolm Masten, M.D., 1051 R Street, Fresno.

Sacramento Valley Counties, June 30 and July 1, in cooperation with Stanford University School of Medicine, Tahoe Tavern, Lake Tahoe. *Chairman:* Joel T. Janvier, M.D., 3632 Marysville Road, Del Paso Heights.

*Chartered buses for Registrants will leave the Ambassador Hotel one hour before the beginning of each course and will return just after each course is over.

AUDIO-DIGEST FOUNDATION

A nonprofit subsidiary of the C.M.A., offers (on a subscription basis) a series of six different hour-long tape recordings covering general practice, surgery, internal medicine, obstetrics and gynecology, pediatrics and anesthesiology. Designed to keep physicians posted on what is new and important in their respective fields, these programs survey current national and international literature of interest and contain selected highlights of on-the-spot recordings of national scientific meetings, panel discussions, symposia, and individual lectures. For information contact Mr. Claron L. Oakley, Editor, 1919 Wilshire Blvd., Los Angeles 57, HUbbard 3-3451.

Medical Dates Bulletin

FEBRUARY MEETINGS

Los Angeles Society of Neurology and Psychiatry in cooperation with California Spinal Cord Research Foundation, Conference "Recent Contributions of Basic Research to Paraplegia." February 17 and 18. Los Angeles. *Contact:* Robert P. Sedgwick, M.D., secretary-treasurer, 2010 Wilshire Blvd., Los Angeles 57.

California Trudeau Society in cooperation with Veterans Administration and Stanford University School of Medicine, Symposium on Pulmonary Disease. February 18, 9:30 a.m. to 4:30 p.m., New Veterans Administration Hospital, Palo Alto. *Contact:* R. Morton Manson, M.D., chairman, 130 Hayes St., San Francisco 2.

California Tuberculosis and Health Association, California Trudeau Society Annual Joint Meeting. February 19 through 22, Jack Tar Hotel, San Francisco. *Contact:* Executive director, C.T.H.A., 130 Hayes Street, San Francisco.

Southern California Society of Gastroenterology. "Problems and Pitfalls in Differential Diagnosis of Jaundice"—Leon Schiff, M.D., February 28, Los Angeles County Medical Association. *Contact:* William E. Molle, M.D., secretary-treasurer, 6221 Wilshire Blvd., Los Angeles 48.

MARCH MEETINGS

Second Low-Beer Memorial Lecture. University of California School of Medicine. March 2, 8:00 p.m. Auditorium-S, Medical Sciences Bldg., U. C. San Francisco. *Contact:* F. Buschke, M.D., Professor of Radiology, University of California Medical Center, San Francisco 22, Calif.

Southwestern Pediatric Society Postgraduate Lecture Series. March 7 and 8, Statler Hotel, Los Angeles. *Contact:* Harry O. Ryan, M.D., secretary, 194 N. El Molino, Pasadena.

Orange County Heart Association, Annual Symposium on Heart Disease. Saturday, March 11. All day. Charterhouse Hotel, Anaheim. *Contact:* Howard G. Buswell, Exec. Director, P. O. Box 1704, Santa Ana.

Anesthesia Section of Los Angeles County Medical Association 6th Annual Spring Postgraduate Meeting. March 11 and 12. Statler Hilton Hotel, Los Angeles. *Contact:* Thomas W. McIntosh, M.D., 686 East Union Street, Pasadena.

College of Medical Evangelists Annual Alumni Postgraduate Convention. Scientific Assembly, Ambassador Hotel, March 14, 15 and 16. *Contact:* F. Harriman Jones, M.D., general chairman, College of Medical Evangelists, 316 North Bailey Street, Los Angeles 33.

Pioneers Memorial Hospital 11th Annual Medical and Surgical Postgraduate Assembly. March 17 and 18. Pioneers Memorial Hospital, Brawley. *Contact:* George C. Holleran, M.D., program chairman, P. O. Box 159, Brawley.

Third Annual Cancer Seminar presented by the American Cancer Society, Nevada Division, Inc., Reno. To be held at the Riverside Hotel Garden Room, Reno, March 28 to 30. *Contact:* American Cancer Society, Nevada Division, Inc., 101 W. Arroyo Street, Reno, Nevada.

Fifth Annual Postgraduate Symposium on Heart Disease sponsored by San Mateo and Santa Clara County Heart Associations, March 29, Veterans' Administration Hospital, 3801 Junipero Serra Blvd., Palo Alto, 9:00 a.m. to 5:30 p.m. *Contact:* John S. Blum, executive director, San Mateo County Heart Association, 45 North B Street, San Mateo, or Mr. William Allayaud, executive director, Santa Clara Heart Association, 461 Porter Building, San Jose.

APRIL MEETINGS

Industrial Medical Association. Biltmore Hotel, Los Angeles, April 11 through 13. *Contact:* Leonard Arling, M.D., secretary, The Northwest Industrial Clinic, 3101 University Avenue, S.E., Minneapolis 14.

Fourth Annual Palo Alto Clinic Symposium sponsored by the Clinic and the Palo Alto Medical Research Foundation, April 15, 300 Homer Avenue, Palo Alto. *Contact:* John F. Weigen, M.D., program chairman, Palo Alto Clinic, 300 Homer Avenue, Palo Alto.

California Medical Association Annual Meeting, Ambassador Hotel, Los Angeles. April 30 through May 3. *Contact:* John Hunton, executive secretary, 693 Sutter Street, San Francisco 2; or Ed Clancy, director of public relations, 2975 Wilshire Blvd., Los Angeles 5.

Pacific Coast Oto-Ophthalmological Society Annual Meeting. April 30-May 4. Riviera Hotel, Palm Springs. *Contact:* Al Miller, M.D., Secretary, 500 South Lucas Ave., Los Angeles 17.

MAY MEETINGS

Los Angeles County Heart Association Annual Meeting, May 3, 12:00 noon to 2:00 p.m. Pacific Ballroom, Statler Hilton Hotel, Los Angeles. *Contact:* Mrs. Sally Smalley, Public Information Director, 2405 W. 8th Street, Los Angeles 57.

California Conference of Local Health Officers Semi-Annual Meeting, May 4 and 5, Los Angeles. *Contact:* Donald G. Davy, M.D., State Department of Public Health, 2151 Berkeley Way, Berkeley 4.

Hawaii Medical Association Annual Meeting. May 4-7. Honolulu, Hawaii. *Contact:* Lee McCaslin, Executive Secretary, 510 So. Beretania, Honolulu 13.

American Association of Genito-Urinary Surgeons (for members and invited guests). May 10-12. Del Monte Lodge, Pebble Beach. *Contact:* William J. Engel, M.D., Secretary-Treasurer, Cleveland Clinic, 2020 E. 93rd St., Cleveland 6, Ohio.

NEVADA CHAPTER AMERICAN ACADEMY OF GENERAL PRACTICE. May 18-20, Riverside Hotel, Reno, Nevada. *Contact:* John M. Watson, Secretary, 1845 Prater Way, Sparks, Nevada.

MEDICAL STAFF OF CHILDREN'S HOSPITAL OF THE EAST BAY Ninth Annual Clifford Sweet Seminar. May 18, 19 and 20. Hotel Claremont, Berkeley, and Children's Hospital of the East Bay. *Contact:* Seymour J. Harris, M.D., chairman, Lectureship Committee, 401 29th Street, Oakland 9.

CALIFORNIA HEART ASSOCIATION Annual Meeting and Scientific Session. May 19 through 21, Disneyland Hotel, Anaheim. *Contact:* J. Keith Thwaites, Exec. Director 1370 Mission Street, San Francisco.

AMERICAN ORTHOPAEDIC ASSOCIATION (members and guests). May 22-25. The Ahwahnee Hotel, Yosemite. *Contact:* Lee Ramsay Straub, M.D., Secretary, 535 E. 70th St., New York 21.

AMERICAN UROLOGICAL ASSOCIATION, INC. May 22-25. Biltmore Hotel, Los Angeles. *Contact:* Mr. William P. Didusch, Executive Secretary, 1120 N. Charles St., Baltimore 1.

MEMORIAL HOSPITAL OF LONG BEACH, Third Annual Medical Staff Symposium. May 24. New Memorial Hospital, 2801 Atlantic Ave., Long Beach 6. *Contact:* George X. Trimble, M.D., secretary, Memorial Hospital of Long Beach.

SUMMER AND FALL MEETINGS

WESTERN BRANCH, AMERICAN PUBLIC HEALTH ASSOCIATION Annual Meeting (joint with U. S.-Mexico Border Public Health Association). June 26 through 29. El Cortez Hotel, San Diego. *Contact:* Robert E. Mytinger, M.P.H., director, Executive Office Western Branch, APHA, 693 Sutter Street, San Francisco 2.

NEVADA STATE MEDICAL ASSOCIATION 58th Annual Meeting and 11th Annual Conference of the Reno Surgical Society. August 23-26. Reno, Nevada. *Contact:* Mr. Nelson B. Neff, Exec. Secretary, Nevada State Medical Association, 506 Humboldt St., Reno.

WASHINGTON STATE MEDICAL ASSOCIATION Annual Convention. September 17-20. Olympic Hotel, Seattle, Wash. *Contact:* R. W. Neill, 1309 7th Ave., Seattle.

WESTERN INDUSTRIAL MEDICAL ASSOCIATION Western Occupational Health Conference, October 6 and 7, Biltmore Hotel, Los Angeles. *Contact:* B. M. Brundage, M.D., Medical Director, Atomics International, P. O. Box 309, Canoga Park, Calif.

LOS ANGELES COUNTY HEART ASSOCIATION Professional Symposium. October 11-12. 9 a.m.—5 p.m., Statler Hilton Hotel, Los Angeles. *Contact:* Manuel Siegel, Program Director, 2405 W. 8th St., Los Angeles 57.

CALIFORNIA ACADEMY OF GENERAL PRACTICE 1961 Scientific Assembly. October 15-18. Statler Hilton Hotel, Los Angeles. *Contact:* William W. Rogers, Exec. Secretary, 461 Market Street, San Francisco 5.

AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC., October 22 to 27, Statler Hilton, Los Angeles. *Contact:* Mr. John W. Andes, executive secretary, 515 Busse Highway, Park Ridge, Illinois.

AMERICAN COLLEGE OF CHEST PHYSICIANS Seventh Annual Postgraduate Course on Diseases of the Chest, December 4 to 8, 9 to 5 daily, Statler Hilton Hotel, Los Angeles. *Contact:* Mr. Murray Kornfeld, executive director, 112 East Chestnut Street, Chicago 11, Illinois.

THE PHYSICIAN'S *Bookshelf*

NINE MONTHS TO GO—Robert McNair Mitchell, M.D., with Ted Klein. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa., 1960. 224 pages, $3.95.

This instruction book for expectant mothers was conceived by a journalist who does medical writing for laymen and he was assisted in its preparation by a Philadelphia obstetrician. It covers the usual ground, from pregnancy tests to layettes, plus some consideration of the economics of obstetrical care (at least in the Philadelphia area) and also advice about subsequent control of conception. A short appendix presents various religious views on birth control, and another appendix provides answers to what are said to be the fifty questions most often asked by prenatal patients. Not all obstetricians would agree with all the answers but, in the main, the advice and information offered seems sound and up to date.

This little volume tries to take a very practical look at obstetric care but, although well written, does not appear to be superior to several other prenatal guides for laymen. We have been treated to several chatty efforts of this sort since it became obvious that Eastman's "Expectant Motherhood" was beginning to rival the Bible as a best seller, and that the market for such books was practically boundless.

CHAS. E. MCLENNAN, M.D.

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PSYCHOPHYSIOLOGIC APPROACH IN MEDICAL PRACTICE—William W. Schottstaedt, M.D., Associate Professor, Department of Preventive Medicine and Public Health, Department of Medicine, and Department of Psychiatry, Neurology and the Behavioral Sciences, The University of Oklahoma Medical Center. The Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11, 1960. 352 pages, $8.00.

This is not another textbook of psychosomatic medicine or a psychology of the practice of medicine written for nonpsychiatric physicians. It is an attempt to define stress in terms of psychophysiological reactions with physical, biological and social parameters and to show how stress works in the etiology of disease processes and how it interferes with healing and restitutive phenomena.

The author clearly shows that stress is an individual matter and that it depends on how an individual evaluates the stimuli impinging on him in terms of his expectations and his past life experiences. His approach is adaptational and not tied to any of the "schools" of psychology. He discusses mental mechanisms as methods for coping with stress and having concomitant physiological expressions which are important in medicine.

The second half of the book deals with interviewing, the doctor-patient relationship and psychological principles of patient management and therapy. Interviewing is presented as a means of identifying and evaluating the stressful life situations which confront a patient. The author emphasizes the importance of the doctor-patient relationship in helping the patient to cope with his stresses. The forces which the patient brings with him and which tend to disrupt the doctor-patient relationship by activating conflicts and anxieties within the physician are discussed in some detail. Methods for coping with these problems are outlined.

This book is a synthesis of a large body of contemporary literature and will save the student many hours of reading and many more of thinking. For the most part it is easy reading. There are some sections which are somewhat nebulous and with somewhat tenuous connection to other sections of the book. However, this is probably more a reflection of the state of our knowledge than anything else. My overall impression is quite favorable and I recommend this book to medical students and the nonpsychiatric physician.

EDWARD J. KOLLAR, M.D.

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INTRASPINAL TUMORS OF CHILDHOOD—Robert W. Rand, M.D., Ph.D., Assistant Professor of Neurological Surgery, University of California School of Medicine, Los Angeles; and Carl W. Rand, M.D., Emeritus Professor of Neurological Surgery, University of Southern California School of Medicine, Los Angeles. Charles C. Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Ill., 1960. 560 pages, $16.50.

In this monograph the authors, father and son, have developed an impressive survey of present-day knowledge of the spinal tumors occurring under the age of 15, using their own series of 72 cases as a point of departure rather than as the principal theme of the discussion. As a result, this book has something of value for anyone having occasion to be informed on matters pertaining to the subject, including particularly the neurologist, neurological surgeon, pediatrician, pathologist and radiologist. The general practitioner with a problem or a bent in the direction of the conditions suggested by the title will also find it profitable. The well planned summaries and tables accompanying most of the chapters facilitate its use as a quick and ready basis of review.

The subject matter is divided with the various categories of disease recognized as basically occupying space within the spinal canal and as such affecting the nervous system. Included are seedings from cerebral tumors as well as those spreading from adjacent structures. The granulomas are discussed, though less extensively than the others. The inclusion of instances of protrusion of intervertebral discs into the spinal canal is neat and pertinent.

Written as it is along the classic format with detailed case reports and an informative review of the literature, without pretense at exhaustive completeness, it also makes pleasant reading beyond its scientific value.

EDWIN B. BOLDREY, M.D.